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SOME DIFFICULTIES MET WITH IN THE PATHOLOGICAL DIAGNOSES OF ENCEPHALITIS.[1]

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To the innumerable perplexing problems confronting the physician in connexion with diseases of the nervous system *encephalitis lethargica* has brought still more and, moreover, adds insult to injury in that death after the most stormy symptoms may, nay, often does, reveal a brain devoid of both naked eye and histological evidence of the specific affection.

Most of us probably served our apprenticeship in the pathology of encephalitis in studies on subjects dead of general paralysis of the insane and only by degrees recognized that quite similar histological pictures might be found in African sleeping sickness, polio-encephalitis and other affections. Nevertheless, it came quite as a shock to me when I found many of the cardinal histological signs of general paralysis of the insane in the brain and cord of a young child dead of so-called *X* disease of Queensland after only six weeks' illness as long ago as 1917.(1) In this instance the typical lesions of anterior poliomyelitis in the small portion of cervical cord available gave the clue to what further lesions might be found within the cranium. Since that date possibly seventy brains with a provisional diagnosis of *encephalitis lethargica* have been submitted to the Mental Hospitals Laboratory for confirmation. In the earlier part of this period Dr. Keith Inglis kept bringing us paraffin hæmatoxylin-eosin sections of the cortex of soldiers dead of this disease in the Randwick Military Hospital and we agreed that some better method of preparation of sections was desirable.

Microscopical Technique.

Brain tissue, especially when pathological and œdematous, does not lend itself readily to paraffin infiltration. Frozen sections were therefore prepared and stained by hæmatoxylin and eosin; they revealed the characteristic mononuclear infiltration of the *pia*, when present, arachnoid congestion and hæmorrhage and any perivascular hæmorrhage or lymphorrhages (cuffing) in the brain itself. But it was desired to reveal more delicate changes in the glia and nerve cells themselves. The routine neuroglia methods in vogue were most uncertain and, moreover, both needed special fixatives apart from formalin and also were not particularly useful for nerve cells or endothelium. Formalin tissue was therefore placed in 70% alcohol for several days, cut in frozen dextrin, affixed to a large slide by celloidin and the whole left in Nissl's methyl blue stain for twelve hours. On two such slides might be placed sections from the Rolandic, frontal, occipital, hippocampal areas and from the optic thalamus, caudate and lenticular nuclei, as well as pieces from the stem including the *iter*, red nucleus and *nigra*, the mid-medulla, cord, if available, and *pons* beneath the fourth ventricle. The slides were dehydrated in strong alcohol leaving an excess of stain behind, blotted, flooded with Ford Robertson's turpentine benzole and slowly warmed on a copper tray over a minute flame. This cooking process is the whole secret and is not easy. When successful and after the influence of time, sunlight and the action of certain properties in the Canada balsam, the nerve cells stand out undistorted and I had some hope of diagnosing an unhealthy neurone.

But above all subpial felting, the various mononuclear cells of the *pia*, the larger glial cells as well as the finest glial fibres and early rod cells showed up readily. At the same time I always had similar pieces of tissue taken past absolute alcohol to cedar wood oil and legroin at times with the extra steps of absolute alcohol, alcohol-ether, celloidin and absolute alcohol and chloroform, then to the cedar oil-legroin and paraffin. Stained by hæmatoxylin and eosin these sections proved quite useful and when well infiltrated gave valuable Nissl specimens. Weigert-Pal and Busch-Marchi sections of the cord were useful additions. It goes without saying that apart from unequal fixing and staining, one section might reveal definite lesions quite invisible in others and this specially applied to neurological peculiarities. As these thick sections, up to twenty or twenty-five microns thick, absorb a considerable amount of light, a good optical system is desirable. This will be met by an apochromatic eight millimetre or one-third inch lens of numerical aperture 0·65 which will show much more than the ordinary one-sixth inch. As the better quality one-sixth inch lenses will not resolve through a thick coverglass, an oil immersion one-seventh inch lens is useful here. This eight millimetre lens readily stands a compensating eyepiece with a twelvefold magnification. An achromatic substage condenser with the best critical light and achromatic bull's eye condenser complete the system. Under such conditions a picture of the cortex is something quite different from an ordinary paraffin preparation.

The Histological Lesions.

If one looks up *encephalitis lethargica* in a standard text book, one reads: "In the early stages the cherry red surface of the brain showing numerous subarachnoid hæmorrhages and on section the purplish-red grey matter and prominent blood vessels are almost pathognomonic."(2) Unfortunately this picture is true of many brains after a congestive attack and while it may be very easy to demonstrate this picture and certain histological lesions at a *post mortem* examination of a patient with *encephalitis lethargica* whom the pathologist has watched for weeks or months, quite another problem confronts him when he receives the most meagre history including neither age nor biochemical reactions of the blood or cerebro-spinal fluid and perhaps only a piece of badly fixed cortex

[1] Being a paper read at a demonstration held by the Section of Neurology and Psychiatry of the Australasian Medical Congress (British Medical Association), Sydney, 1929.

when the typical lesions are supposed to lie in the brain stem.

In the text books microscopical changes are also noted, such as hæmorrhages and lymphorrhages into *pia mater* and perivascular spaces, even larger hæmorrhages in the medulla, *pons* and brain stem. In time there follow definite neuroglial reaction, mesoblastic proliferations, softenings, alterations, some temporary, some permanent, in the nerve cells themselves, leading to their ultimate disappearance. Opinions are divided as to whether the neurones sustain real damage before any other system or the reverse.

More unanimous are the opinions that in *encephalitis lethargica* the main brunt falls on the brain stem and central nuclei, as in poliomyelitis the cord is the place of election. Put another way, Dieterle reminds us that the outer and inner zones of the cortex are relatively avascular and the peculiar anatomical features of the long and short cortical vessels from the *pia* may explain certain peculiar features in general paralysis of the insane. His thick sections give a good bird's eye view of a capillary reaction in this and I think other types of encephalitis. On the other hand the areas supplied by the ganglionic system to the basal nuclei are the more often affected in *encephalitis lethargica.*

Malamud and Lowenberg[4] were fortunate enough to obtain the brain of a patient in the very early stage of general paralysis of the insane. He was almost symptom free and dead of an accident. The Bordet-Wassermann serum response was positive. Definite changes were present in the aorta. I must give the histological description of his lesions, because in quite a number of similar examinations carried out by myself after first returning to Australia there was the same paucity of reaction or earliness of lesion compared with material used by Ford Robertson for typical reactions in the brains of general paralytics in Scotland.

> The leptomeninges were generally delicate, in places, however, somewhat thickened with connective tissue proliferation. There were recent superficial hæmorrhages in the *pia.* A mild but distinct meningitis obtained with lymphocytes and plasma cells, but not polyblasts in the pial spaces and adventitia of cortical vessels. The cortex showed no sign of inflammatory reaction. The architecture was perfectly intact. Other cortical vessels showed no infiltrations and the glia was not affected. The white matter was even less affected.

Now this picture reveals nothing specific and if a brain of a person with a history typical of *encephalitis lethargica* showed as much, I should consider myself fortunate. Malamud describes successive stages of reaction as do other workers and still the picture would do for other encephalitides. As a matter of fact further inquiries into the histories of some patients whose brains were sent to us with the diagnosis of *encephalitis lethargica,* almost certainly suggested a syphilitic ætiology; our task was rather first to decide whether any lesion was present and then to rule out syphilis. With this object the patient's age and history were noted. Since other morbid states, even non-nervous diseases such as nephritis, may cause some reaction in the central nervous system, a good history which includes these, is requisite. Such a conception as a normal brain is not a fixed standard picture. At least specimens presumed to be normal have on section shown definite lesions. Gliosis, however, according to Ford Robertson is extremely rare apart from a definite neurological or mental case. In my series a sudden onset was noted as common. Stevenson[5] encountered 71 sudden onsets in a series of 83, but this also is not unknown in general paralysis of the insane. The absence of reaction to the Bordet-Wassermann test with serum and cerebro-spinal fluid, to the cell globulin and gold sol tests and to other tests, too, was obviously a help.

The duration of the illness might indicate what period or stage of lesion to look for. An effort was made to ascertain if these biological findings as well as certain phenomena usually considered pathognostic of cerebral syphilis would allow the picking out of this disease with certainty. Such phenomena would include plaques of endothelial proliferation, chronic interstitial thickening and dense subpial felting, definite granulations (gliosis) on the ventricles, such advanced perivascular infiltration and proliferation as to include an extensive mesoblastic reaction and areas of sclerosis with cortical disorientation and neurone loss, together with mononuclear infiltration of the *pia mater* of the cord like that of the cortex and posterior spinal ganglia and nerve roots, with perhaps a tract lesion in the posterior columns. Experience showed the necessity of utilizing both methods of approach. Histology alone was not to be depended on. At the best one often had to be content with indicating the presence of an encephalitis which might be of such and such a type.

Clinical Differentiation.

Ives Hendrick[6] writes:

> A history of encephalitis has been considered authentic when the diagnosis was made during previous hospitalization at a first class institution, or when there has been an illness of several months, with such characteristic features as delirium or *lethargica,* neurological changes such as diplopia, tremor, transitory paralyses, choreiform movements, delusions or hallucinations.

And I may add the performance of the above and other biochemical tests. Of the pathology he says:

> Convincing evidence of the persistence of the virus after the acute stage is passed, is being presented.

While I admit that many of the above signs and symptoms were common in my series, the absence of a regular series of efficient clinical notes and associated useful material (the special committee of the Medical Research Council demands material not more than six hours after death) precluded me from carrying out my original intention specially to describe *encephalitis lethargica.* Instead I have had to present the histological findings in typical groups of affection associated with a history of *encephalitis lethargica* and in material submitted for confirmation of the diagnosis.

Analysis of the Cases.

Of the seventy cases roughly twenty-nine showed gross encephalitic lesions; in a further twelve the signs were moderate but definite, while in fourteen more such signs as were present, were not sufficiently specific. It was not possible to prove that the signs might not have been caused by some agency other than a living organism, such as lead or food poisoning. The remainder either revealed no changes or had not been in a suitable condition or efficiently fixed for sectioning.

Again speaking roughly, it would appear that the *pia*-arachnoid seemed most affected in seventeen, in four or five so much so that the changes were macroscopic. Large glia cells and subpial felting in the first cortical layer seemed the chief interest in twenty-eight specimens, some sclerosis or cell destruction in the substantia in five and similar neurone destruction, central nuclei in three or four. It is with diffidence that I would diagnose with certainty as of importance slight neurone changes in the brain after having read Crile's work. Lesions of the *cornu Ammonis* seemed to have been defined in six specimens, of the cerebellum in eight, of the spinal cord in eight and of the olives in at least three. No specimens from persons in whom the diagnosis of general paralysis of the insane had been made before death were included, of course, but in some instances this diagnosis had to be made after a *post mortem* examination.

Many patients die of *encephalitis lethargica* with little to show apart from deep congestion and petechial hæmorrhages in the *pia*, cortex and brain stem. A long search may reveal a few mononuclear cells where one would not expect them normally. Pathologists are warned against attaching too much importance to excessive hyperæmia of the brain *post mortem*. Temple Fay[7] who happened to be reflecting the *dura mater* over the motor area, may be followed. No focal lesion was found, but a milky translucent arachnoid distended with fluid, a generalized arachnoiditis; a few moments later, so intense was the congestion, he feared he had ruptured a pial vessel. Meanwhile the anæsthetist reported the patient was having a convulsion.

This vascular picture may pervade the brain and stem to a great degree and may be all that the pathologist can report. At times, of course, a definite hæmorrhage may be found under the ventricle in the *pons* or stem.

I had the good fortune to be able to follow clinically a patient in one of the mental hospitals and to perform the requisite biochemical tests. The case was reported in THE MEDICAL JOURNAL OF AUSTRALIA, July 28, 1923.[8] Syphilis and septic meningitis being excluded, the presence of a brain reaction including infiltration of the *pia* and adventitial sheaths of the vascular system throughout the cranial contents with mesoblastic reactions near the blood vessels as well as the earliest signs of neuroglial reactions (rod-shaped nuclei and so on) allowed a definite diagnosis.

A woman, aged thirty-seven, whose case was reported in THE MEDICAL JOURNAL OF AUSTRALIA of January 22, 1927, while at work and apparently quite well, collapsed, became unconscious and died within twenty-four hours. The above signs as well as a hæmorrhage into the occipital pole and equally full biochemical tests led to a correct diagnosis. That there was more advanced gliosis in this specimen reminds me that the disease must have been present some time and have caused no apparent symptoms and the neurones must have been functioning. As in syphilis, therefore, there need be no real parallelism between the intensity of the inflammation and the parenchymatous changes. The changes in many of the specimens corresponded to this degree and it is obvious that the final diagnosis must lie with the clinician.

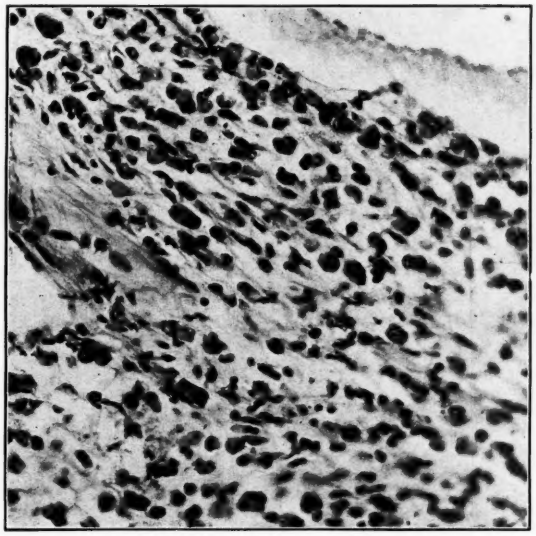
Hadfield,[9] speaking of encephalomyelitis, stated:

> There is a need of defining accurately the histological picture and the differentiating from other types of encephalitis. . . . Perhaps this will be found in the minute study of the cellular infiltration and the nature of the microglial reaction.

The next specimen was the brain of a returned soldier who had been a patient at the Randwick Military Hospital. He had had influenza in England and had been fourteen days in hospital; on the ship coming out he had been ill for five days with meningitis; during two months since he had been "dopey." His knee jerks were exaggerated. His pupil reactions were normal. His skull had been trephined and his brain tended to herniate. The Wassermann test with his serum did not yield a reaction. The original diagnosis was cerebral abscess, but nothing being found *post mortem*, *encephalitis lethargica* was considered.

The *pia*-arachnoid was thickened, œdematous, hæmorrhagic and infiltrated with mononuclear cells. In the first layer of the cortex there was slight but definite subpial felting; everywhere there was increase in the number and size of the glia cells; the blood vessels were engorged, but there was little else abnormal. In the deep layer of the cortex there was intense congestion with numerous petechial hæmorrhages, some neuronophagia, the larger cells being better preserved than the others. Lines of lymphocytes and plasma cells indicated cuffing of the capillaries. Areas of friable, œdematous tissue surrounded the larger vessels where phagocytic cells filled with lipoid pigments or hæmatoidin were found. Innumerable glia cells, too, were noted. The lenticular nucleus seemed specially prominent by reason of many almost naked eye hæmorrhages. A peculiar change in some of the endothelial cells was noted; becoming amorphous they formed the starting point of calcarious deposits[10] about the vessel walls (Russell's colloidal plasma cells).[11] Doubtless it is more often the plasma cells which undergo this degeneration, but I have been successful in tracing the change in the endothelial cells of blood vessels when there was great neuroglial proliferation and hardly any other type of cells. The change can be observed in both brain and peritoneal tumours and in the meninges. Besides, numerous pycnotic granules becoming calcarious were observed in the perivascular spaces. Early calcarious changes are a feature of both the

ILLUSTRATIONS TO THE ARTICLE BY DR. OLIVER LATHAM.



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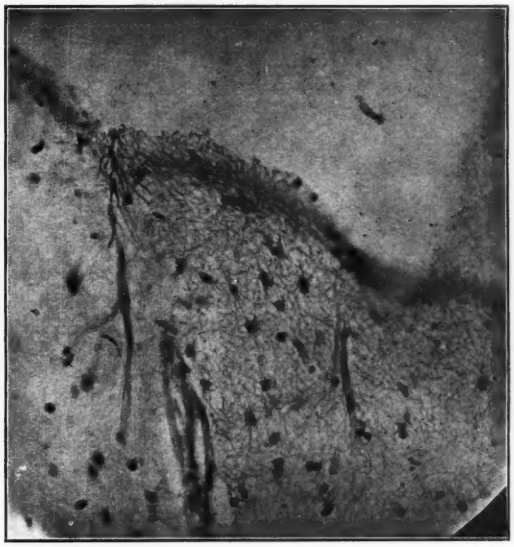

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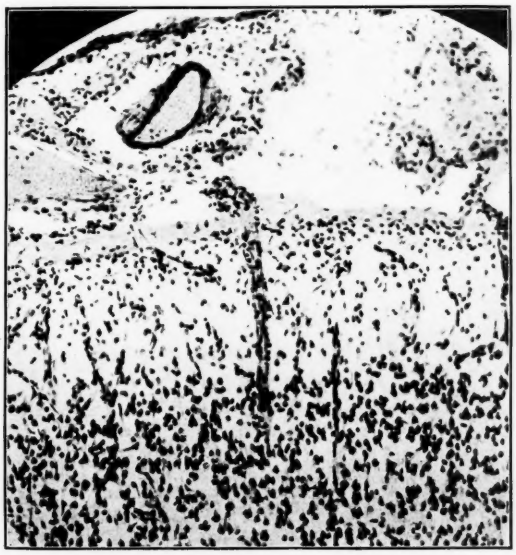

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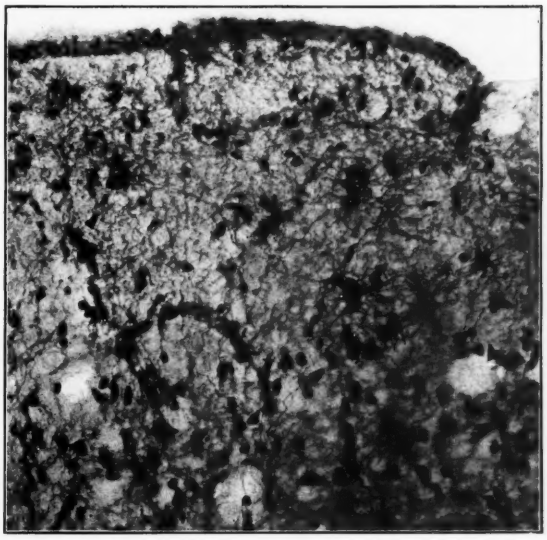

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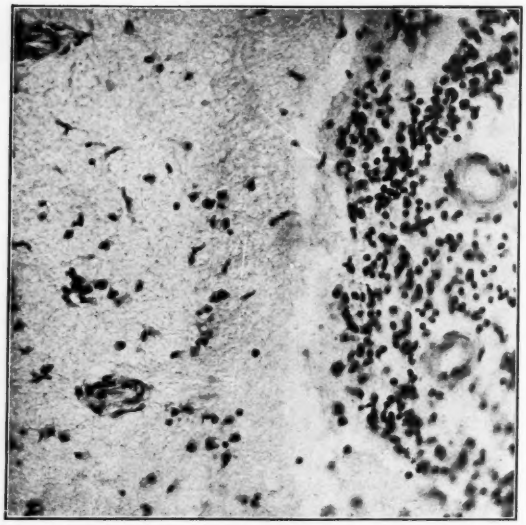

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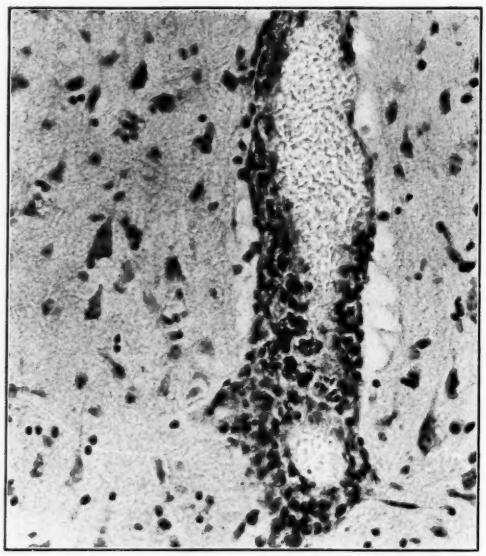

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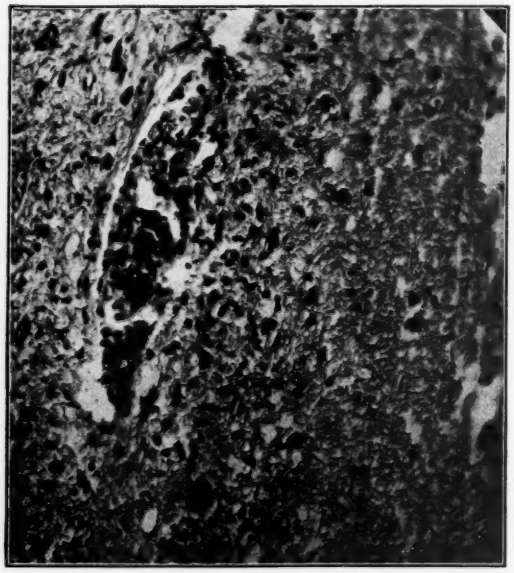

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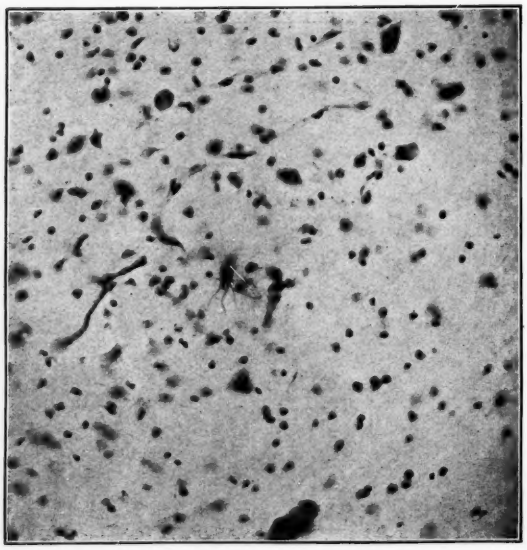

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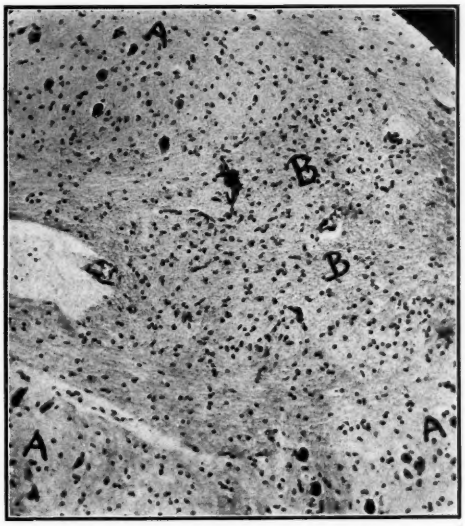

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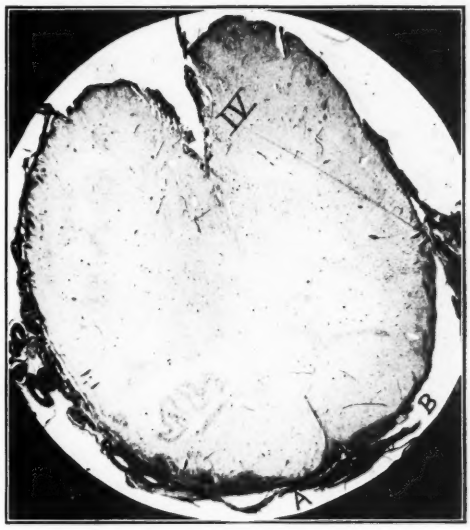

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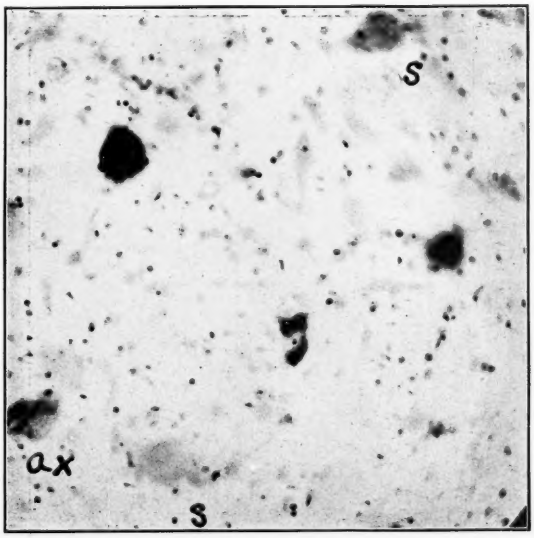

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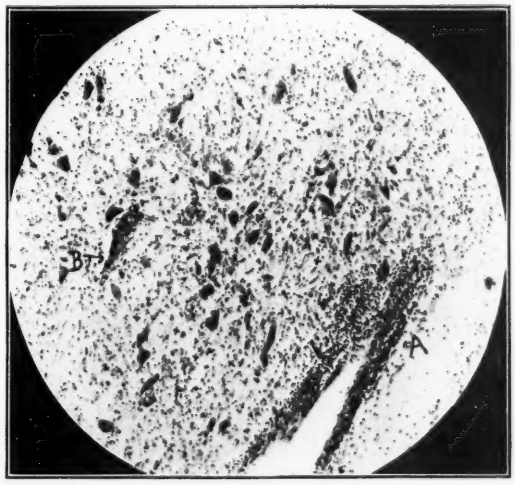

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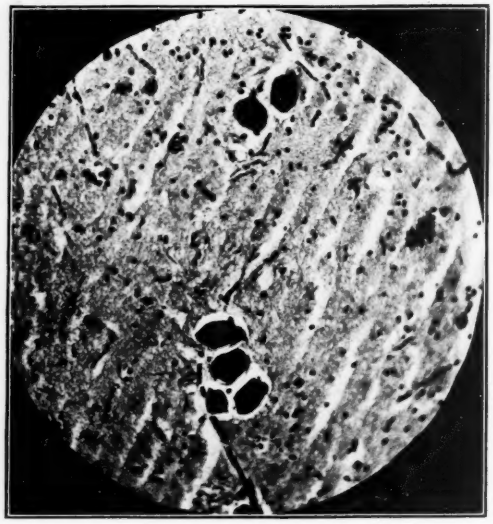

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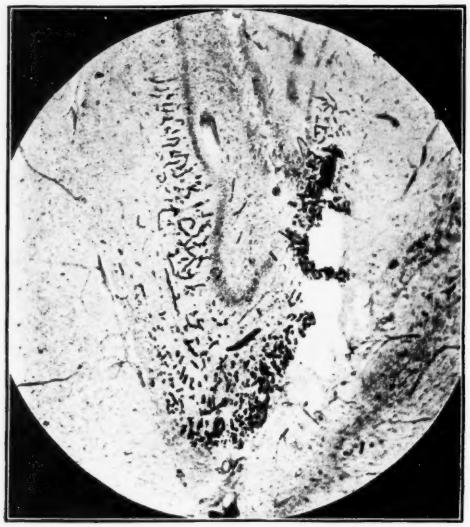

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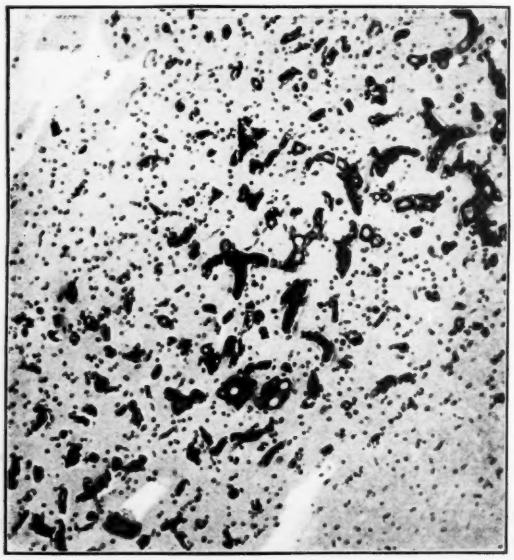

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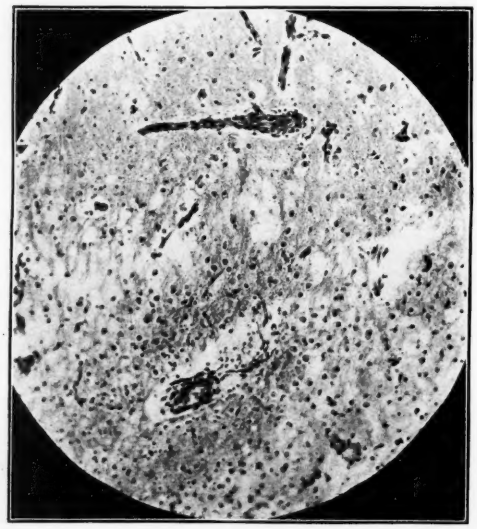

FIGURE XVI.

natural and experimental encephalitis in animals. It was noted that whenever these changes were worst severely necrosed nerve cells, some hyaline cells and some cells of almost pure lime were present.

As many medical practitioners do not often see many patients with *encephalitis lethargica*, difficulties in diagnosis are bound to occur. Bodies are referred to us from the public morgue after sudden death with no discoverable lesions *post mortem*. Case XXVIII-46 was referred to me with a history of a long exhibition of "606." The tissues unfortunately were mushy and poorly fixed, yet a definite degree of reaction which might denote general paralysis, was present. On the other hand, very evident were little areas of unorganized cellular reactions near the vessels (Jakob's unorganized granulomata).[11]

Case XXVIII-48, received the same day from the morgue, was more to the purpose. The patient had been under Dr. Susman's care seven years before for encephalitis. There had been definite Parkinsonianism; he had had a fixed and rigid gait and had been quite an invalid. Death had taken place by drowning in a shallow pool.

Post mortem some doubtful degenerated areas were found under the frontal lobe, possibly the result of an old injury. Histological examination revealed a fibrous and thickened *pia* with numerous endothelial plaques on the *pia* of both cerebrum and cerebellum, usually associated with general paralysis of the insane. There was some possible felting and doubtful gliosis of the cortex, definite cuffing and neuronophagia in the deep layers of the cortex, but of more interest was the gliosis of the white matter of the cerebellum.

Case XXVIII-18, kindly sent by Dr. Dawes, of Orange. A man, aged forty years, fell off a low building; he was unconscious for about half an hour, was admitted to hospital, went home and returned in a few weeks with headache, nystagmus and giddiness. The pressure of the cerebro-spinal fluid was not increased and the gold sol test did not yield a reaction. The cells and the globulin contents of the fluid were twice found to be normal. The Bordet-Wassermann test did not yield a reaction. He wandered away from the hospital, was taken back and died in a day, irrational up to the end. The *post mortem* examination revealed a healed fracture of the eighth and ninth costal cartilages. All his organs were normal; there was no excess of the cerebro-spinal fluid and the brain was not even congested. The skull and vertebræ were normal and there were no adhesions between the membranes and the brain nor signs of old hæmorrhage.

The ultimate clinical diagnosis lay between *encephalitis lethargica* and chromatolysis following injury. I propose to discuss the latter idea later, but wish to emphasize the value of all the collateral information available. Only a piece of the cerebral and cerebellar cortex was received. It was found to be slightly hyperæmic. There was a slight excess of the mononuclear cells in the *pia*, slight cuffing and perivascular hæmorrhages with definite toxic swelling of the vessels and endarteritis, but the outstanding feature was the number and size of the glia cells, a few in the molecular layer, but the majority in the white matter of the two cortices. But for the biological data I might have suggested syphilis.

Two equally excellent neurological and biochemical histories were received with two brains handed in by Dr. S. A. Smith. Both patients had eye symptoms.

The first, aged forty-eight, had diplopia and then ptosis and died after a history of six weeks' illness with an ulcerated cornea. There was no reaction to the Wassermann test with blood serum and no increase in the cell content of the cerebro-spinal fluid. No tubercle bacilli were found and there was no growth on culture. Petechial hæmorrhages, cuffing, neuroglial proliferation and neuronophagia were noted; these were most noticeable in the *pons* beneath the fourth ventricle.

The other patient had a history of eleven days' illness with severe and lasting pain in the right eyeball and then headache, with tenderness of the back of the neck. Parkinsonian tremors and respiratory difficulties appeared and the latter were responsible for his death.

The histological examination revealed that infiltration and reactionary cellular phenomena were at a minimum, yet early but widespread glial reaction was a feature, especially around the blood vessels. Although there was but little cuffing, there was so much toxic swelling that it resembled hyaline changes. In the stem many amyloid bodies were noted and many nerve cells obviously degenerated. Some were mere bags of pigment. In the *pons* beneath the fourth ventricle and especially the *optic thalamus* neuronic destruction could readily be followed. Subpial felting and glial reaction in the first cortical layer were present.

Cases XXIV-31 and XXV-34 are mentioned only because uræmia and alcohol respectively were included as ætiological factors. Signs of encephalitis were recognized microscopically. Douglas McRae reminded me in Morningside that in a small percentage of patients the condition clinically diagnosed as general paralysis of the insane was really due to vascular phenomena in nephritics. This was before the introduction of the Bordet-Wassermann test. I have seen similar references in the literature. Macdonald Critchley[12] discusses "arterio-sclerotic Parkinsonism" and Lhermitte and McAlpine[13] "syphilitic encephalitis of the *corpus striatum*" and quote Wilson and Cobb as having collected a number of cases of *paralysis agitans* accompanying tabes of general paralysis in a paper entitled "*Mesencephalitis Syphilitica*." Possibly the affection in both of these patients was lues. In one specimen there were lesions rather like miliary gumma in the olives and the cortical reactions, too, are suggestive of syphilis. However, the serum Bordet-Wassermann test twice yielded no reaction. The assistance of the pathologist has to be sought in cases of the following type.

Case XXIV-14: A young bank clerk in a northern town was in perfect health; he was suddenly attacked by headaches, mental confusion, coma and died in five days. The diagnosis was *encephalitis lethargica*. No reactions were obtained with his blood to the Wassermann and Kline tests. The Wassermann test applied to the cerebro-spinal fluid failed to yield a reaction. There was no excess of globulin in the cerebro-spinal fluid; the fluid contained 120 cells to each cubic millimetre. A piece of cerebral cortex and a part near the lateral ventricle were secured for examination. There was slight œdema of the *pia mater* with small collections of mononuclear cells scattered throughout the tissue and in these situations definite subpial felting and equally definite increase in the number and hypertrophy of the glia cells. Toxic swelling of the vascular system generally with occasional but definite cuffing and a few giant glia cells in the deeper tissues were noted.

Case XXIV-1: The patient, a woman, suffered from an acute mental disturbance after abortion; the illness was like septicæmia. An alternate diagnosis was *X* disease.

There was considerable thickening and œdema of the *pia mater*, sufficient subpial felting and gliosis not only of the cortex but also of the whole nervous system examined from cortex to cord to suggest general paralysis. There was, however, real destruction of many cells with scattering of pigment in the *substantia nigra* and also in addition

to the gliosis and petechial hæmorrhages acute degenerative rather than reactive vascular phenomena.[14][15] Cuffing was strangely absent.

Encephalitis in Children.

Nine brains and a few cords were received for examination in addition to those with frankly tuberculous lesions and one brain of a man who had died of tetanus. Nearly all of these were definitely pathological. In one instance the diagnosis was *X* disease and in the remainder it was *encephalitis lethargica*.

Case XXIX-25. The last brain of my series was from a patient who was also sent by Dr. Dawes, of Orange. The condition both from a pathological and from a clinical point of view was most acute. A child, about five years old, was apparently quite well till she was seized with a succession of fits and died in twenty-four hours. The history was vague. There was no fever, paralysis or localizing signs and no excess of cerebro-spinal fluid.

Great difficulty was experienced in sectioning this material possibly on account of pathological friability, although it had been placed in formalin within four hours of death and the only lesion of any note was a peculiar, partly fibrous, partly granular thickening of the cortical *pia* in places and the *pia* around the *pons varolii* and brain stem. Considerable hæmorrhage had taken place into the membrane and there was a slight excess of lymphocytes. Careful examination, however, of the hippocampal region with the most critical light revealed innumerable *Stabzellen* with most delicate processes chiefly pointing towards the blood vessels. This enabled me to judge the presence of an early but widespread encephalitis.

The *pia*-arachnoid of some of the other brains was so infiltrated with lymphocytes *et cetera* as to suggest a lepto-meningitis. One of these from Narrandera Professor D. A. Welsh in my absence was kind enough to report on thus:

Outstanding features are the infiltration of the *pia* with mononuclear, even multinuclear cells, with some calcarious particles. Many of the small cerebral blood vessels show toxic thickening of their walls with hæmorrhage. There is considerable underlying gliosis where *pia* is thickest. In the larger masses cells are becoming fibrous, in part caseous. Whether this (chiefly mononuclear) leptomeningitis denotes an *encephalitis lethargica*, a tuberculous or a typhoid process (a positive Widal response had been obtained) cannot be determined, since no midbrain was included.

Space will not permit discussion on the others save one, number XXVIII-11. In the brain of a child, aged three years, among other signs there were definite areas of softening in the cerebellum and brain stem and hippocampus associated with numerous *Gitterzellen*, the various areas being separated or surrounded by large numbers of giant glia cells, especially in the hippocampus; these lesions were associated with the most definite productive endarteritis. The Weigert-Pal and Marchi cord sections were normal. Such a combination of lesions in a young child proved puzzling until I read Winkelman and Eckel's article[16] on productive endarteritis in young people, wherein they picture lesions similar to those of this patient occurring

LEGENDS TO ILLUSTRATIONS.

FIGURE I.

Mononuclear (polyblast) infiltration of pia-arachnoid; from a patient with *encephalitis lethargica* of five weeks' duration. Not unlike a meningeal reaction far removed from a tuberculoma.

FIGURE II.

Same case; subpial felting and glial reaction.

FIGURE III.

Low power view of *pia* and cortex of a patient aged eighteen years with congenital paresis; no symptoms till eighteen months previously. Laboratory modification of Nissl's method.

FIGURE IV.

First cortical layer, general paresis, showing long glial fibres invading the cortex. Ford Robertson's methyl-violet.

FIGURE V.

Typical reaction in the cerebral cortex, in the *pia*, neuroglia and vascular system; from a young woman lethargic for two years. Toxic thickening some vessels and productive arteritis in others.

FIGURE VI.

Obscure encephalitis in a patient whose serum did not react to the Wassermann test; "Mesenchymatous mesh" (unorganized granuloma of Jakob).

FIGURE VII.

Encephalitis lethargica in a soldier; inflammatory foci near a small blood vessel (Wilson) in the pons.

FIGURE VIII.

Early gliosis in the olive; acute encephalitis in patient aged twenty-one (puerperal). Note large glia cell in centre.

FIGURE IX.

More chronic sclerosis in the olive in obscure encephalitis. No response to the Wassermann test. Regular line of nerve cells in the olive at A, A, A, cease abruptly at B, B, owing to inflammatory foci near vessels. Precisely similar conditions may obtain in *X* disease.

FIGURE X.

Generalized encephalitis associated with a basal meningitis (mononuclear). At IV the ventricular ependyma is obviously affected. The third ventricle and aqueduct were also affected.

FIGURE XI.

Neurones beneath the fourth ventricle showing axonal degeneration at AX, chromatolysis and shadow forms at S, S.

FIGURE XII.

Destruction of many cells in the *substantia nigra* with liberation of pigment x, x, x, x, causing smudging of picture. Note mononuclear cuffing of vessel A and at B; early calcification in form of minute granules in vessel wall. Figures XI and XII are from a patient with typical *encephalitis lethargica* of twelve days' duration.

FIGURE XIII.

Nerve cells in the brain stem almost calcarious. There were many other neurones more or less affected. *Encephalitis lethargica* in child, aged 2½ years, at the Royal Prince Alfred Hospital.

FIGURE XIV.

Almost complete calcarious degeneration of the vascular system in hippocampus, in obscure encephalitis.

FIGURE XV.

Calcarious degeneration of the cortical capillaries in patient with chronic plumbism. Cerebral lead content twenty-one times normal.

FIGURE XVI.

So-called productive endarteritis in cerebellum in a child, aged six years, at the Royal Alexandra Hospital for Children, diagnosed *encephalitis lethargica*. Note the remarkable proliferation of endothelium and areas of softening with *Gitterzellen* and patches of large neuroglia cells. Quite similar pictures are seen in food poisoning and artificial encephalitis in animals chemically poisoned.

after food poisoning. Their own illustrations and quotations from Nissl and Alzheimer would tend to show that the so-called *endarteritis syphilitica* of the small cortical blood vessels sometimes present in syphilis and other forms of encephalitis was not really due to the presence of spirochætes and therefore was not specifically syphilitic, but might be the result of other toxins possibly not even bacterial but biochemical or chemical, such as food poisons, lead or the like. The illustrations certainly resemble superficially those from a patient with infective encephalitis. I am indebted to Dr. Macintosh and Dr. Phillis Anderson for this unusual specimen.

The Polio-Encephalitis Group.

A few brain specimens of persons dead of so-called Landry's paralysis with brain symptoms were brought to me. It is stated that most of these "will turn out to be types of poliomyelitis."

Case XXIX-13. A spinal cord received recently from the Western Suburbs Hospital revealed all the histological signs observable in the cords of monkeys artificially inoculated with material from infantile paralysis and I could trace quite severe lesions accompanied by multiple hæmorrhages up into the *substantia nigra* where a number of cells were quite "knocked out" either by hæmorrhage or by those mesoblastic reactions so common in the other encephalitides. In the brain cortex there was found not only a hæmorrhagic *pia*, endarteritis of the small vessels with a little cuffing, but actually an inflammatory reaction with fibrosis among the Betz cells.

McIntosh states: "My own investigations convince me that extensive involvement of the cortex in anterior poliomyelitis is unusual and usually confined to a few scattered areas of perivascular lymphorrhages."

In another patient who had both brain and spinal symptoms, the *post mortem* examination revealed an extensive hæmorrhage into the *cauda equina* and gliosis and axonal degeneration of nerve cells in every level of the cord and cortex. I have mentioned that the cord of the one patient with *X* disease was as much affected as the cortex. In the majority of those with brain affections but little pathological change was found in the cord. Although the patient in Case XXIII-30 was referred to me as suffering from Landry's paralysis by Dr. Lidwell, his condition differed from any other I have previously described in that, besides numerous obviously degenerated neurones which can be counted in the cord, the medulla, the *pons*, the central nuclei and the cortex and the presence of a distinct subpial felting and giant glia cells among the cell groups beneath the fourth ventricle, there is also to be noted a gross myelitis around the periphery of the cord, chiefly over the posterior columns and on either side of one anterior horn. I was reminded by this Busch-Marchi reaction indicating extensive myelin destruction of that class called by McIntosh[17] "encephalo-myelitis" in which the usual signs of poliomyelitis are accompanied by extensive demyelination, chiefly around the vessels and around the periphery of the cord, allied to disseminated sclerosis. This is the type occurring after vaccination with cow pox and is to be sharply differentiated from other classes described.[18]

Traumatic Encephalitis (Armour).

Case XXVIII-43 brings up the vexed question as to whether a cerebral concussion can produce an histological encephalitis.[19][20] In this case a woman fell from a tram; mental symptoms appeared, but not immediately and she died in three weeks. *Post mortem* examination disclosed nothing. Histologically I noted that the *pia* was thickened here and there, especially near the *hippocampus*, with definite subpial felting, gliosis both in the superficial and deep cortex and around the *locus perforatus*, that is, the vessels to the central nuclei, the vessels themselves revealing typical toxic thickening.

I was asked whether there was any pathological evidence of the psychosis.

Authorities are not helpful in regard to the exact histological picture in the contused brains of those with no fracture of the skull, no numerous hæmorrhages and no history of gassing.[21] In many of Mott's patients[22] shell shock was largely dependent on gas effects. Sharpe says of cerebral concussion:[23]

Operation reveals a wet brain with more or less hæmorrhage and later a white hazy connective tissue formation, the residue of the former supracortical hæmorrhage. This connective tissue blocks normal absorption, producing chronic cerebral œdema.

And Armour continues: "This eventually leads to dilatation of the ventricles."

Perhaps all this is true, but it seems difficult to ascribe my findings, typical as they were of a mild encephalitis, to cerebral concussion, when the *post mortem* examination three weeks later revealed no rusty staining or evidence of naked eye reaction. Such widespread reactions indicated, I thought, an encephalitis which might have caused the accident. However, the Court decided that death had been caused by the accident. Following more gross head injuries, I have found quite definite signs of an encephalitis in two returned soldiers, one of whom had fractured his skull and had undergone several operations and the other had a self-inflicted gunshot wound of the brain. In both cases pieces of cerebrum as far removed from the site of the damage as possible were examined. The histories, however, were incomplete. The biological tests might have thrown a great deal of light on the situation. Time and again pathological experience has shown that traumatic lesions have had a syphilitic encephalitis basis.

On two other occasions I have been asked by Dr. Guy Prior and Dr. Keith Inglis respectively to assist in court inquiries into the cause of death. One of the people had a fall of four feet and was found to be affected with transitory paralysis and permanent but slight leg wasting. Two years later the histological findings revealed a syphilitic cerebritis and an old hæmorrhage in the cord together with some tabetic tract degenerations. A Wassermann reaction was obtained. In Dr. Inglis's

patient death revealed the signs of Erb's syphilitic spinal paralysis.

Finally, another threatened court case had to do with the death of an insane patient who had worked for ten years in lead. The first diagnosis of general paralysis of the insane was challenged because the Wassermann test with the blood serum and cerebro-spinal fluid did not yield a reaction. The cell count of the cerebro-spinal fluid was normal, while there was but a trace of globulin and a partial colloidal paraffin reaction was obtained. The provisional diagnosis, therefore, lay between *encephalitis lethargica* and lead poisoning. The finding of large glial cells with fibrous and endothelial thickening of the *pia* and vascular system together with the presence of a little endarteritis and the most extensive calcarious degeneration of whole areas of capillaries with minimum mononuclear reaction as well as some loss of neurones and petechial hæmorrhages tallied with Mott's description[24] rather than with Oliver's. Mott laid emphasis on the paucity of mononuclear cells and the absence of ventricular granulations in lead poisoning. Mr. R. K. Newman, of the Cancer Research Committee, found seven milligrammes of lead in five hundred grammes of brain or about twenty times the normal, a rather unusual finding, since the bones hold nearly all the unexcreted lead.[24]

Summary.

The histological and where possible the biochemical findings in picked groups from some seventy patients whose affection was diagnosed as *encephalitis lethargica*, have been presented.

The basic similarity of the histological lesions in the nervous system of all types of encephalitis has been alluded to.

The great value of the biological findings to both clinician and pathologist is emphatically urged and explained.

Legitimate reasons why the pathologist should have access to the whole brain and spinal cord and some posterior spinal ganglia and peripheral nerves are advanced. Lesions although widespread are often irregular in distribution.

The necessity for the material to be efficiently preserved, fixed within six hours of death and not disorientated by being sliced when soft and unfixed, is noted.

The results obtained may remind clinicians and coroners of the value of the histological examination of the nervous tissue of victims of sudden death or obscure nervous affections, especially when the *post mortem* examination reveals little or nothing.

Acknowledgement.

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NOTES ON UROLOGICAL SURGERY GLEANED ABROAD.[1]

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I HAVE been asked to speak to you tonight of my impressions of the urological work I saw abroad and I thank you for that honour. I propose to take some of the commoner urological conditions and to describe briefly some of the methods commonly used in dealing with these in the clinics I visited.

A word first on diagnosis of urological diseases. One realizes after visiting clinics abroad, particularly in America where they are so lavishly equipped with every convenience, what strides have been made in recent years in improving diagnostic technique. This has been achieved with the help of (i) improvements and developments in the cystoscope, both for diagnosis and modifications to enable many manipulative and operative procedures to be carried out easily; (ii) improved radiological technique and interpretation, a better understanding of pyelography and its uses, cystography and pyeloscopy; (iii) standardization of laboratory tests, particularly those for renal function and their clinical application.

The result is that in this branch of surgery an accurate and complete diagnosis, meaning by that a full knowledge of the condition of the whole urinary tract and its reserve ability to function, can and should be made in practically every case. The necessity for operation of an exploratory nature is eliminated.

These newer methods cannot and should not be dispensed with in order to attain the highest degree of accuracy in diagnosis of lesions of the urinary tract and I want here to insist on the importance of a full and expert investigation of the urinary tract in the following conditions which only too often one still sees let drift along without a proper diagnosis of their cause.

The first is hæmaturia, gross or blood cells in microscopical quantity in the urine, of which the origin is not absolutely certain, even if the condition appears to clear up under simple treatment, as it undoubtedly will in many cases. The chief danger here is that a tumour of the bladder or kidney will be overlooked in perhaps an early and removable stage and give no further evidence of its presence often for months or years.

The second condition is pus cells in the urine (catheter specimen) persisting, of which the origin is not certain. Such might be the only sign of early renal tuberculosis which, if untreated, will spread to other parts of the urinary tract.

In the third place no patient should be allowed to remain with the diagnosis of cystitis till every possible cause is excluded, whether it be a urethral obstruction, a bladder lesion, such as stone, tumour or diverticulum or a primary infection in the kidney, as tuberculosis, pyelonephritis, pyonephrosis *et cetera*.

In the fourth place, in a pyelitis which does not clear up under efficient medicinal treatment in a few weeks, a primary cause in the kidney should be excluded, as stone, tuberculosis *et cetera*.

In the next place in the presence of X ray shadows anywhere in the region of the urinary tract it is necessary to make a full investigation, on the one hand to determine that this shadow is due to a urinary calculus and not to an extraneous source, for example, a gall stone, a calcified mesenteric gland *et cetera*, on the other hand to get further and full information on the state and function of the whole urinary tract. To show a shadow within the renal area is not sufficient evidence to justify immediate operation. As to whether the cause of the shadow is inside the kidney or ureter an opinion can often be given on its appearance and its mobility on respiration, but the passage of an opaque ureteric catheter or a pyelogram and stereoscopic X ray pictures will prove this point beyond doubt. Then one must know the degree of infection, the amount of distension of the kidney pelvis and the efficiency of that kidney. Also, what is more important, the presence, freedom from disease and relative function of the opposite kidney. These points can all be gained by ureteric catheterization and collection of the separated specimens of urine for bacteriological examination and the estimation of urea percentage in each and a pyelogram of the affected side. Finally, in cases of small stone it is necessary to know in what portion of the pelvis or calyces it lies and if any anatomical abnormalities of the renal pelvis exist. This is shown with a pyelogram, as the series of pyelograms I show you illustrates. When this information has been collected, it is known exactly where the stone lies, by what method it can be removed with the least trauma to the kidney, if nephrectomy is required and if it is safe. The whole examination described, including pyelograms, can be done at one time, with one instrumentation and under a local anæsthetic in the urethra or with very sensitive bladders caudal anæsthesia may be used.

A urinary tract investigation should also be made to clear the diagnosis in patients with atypical abdominal symptoms. The frequency with which one sees that the appendix or gall bladder has been removed or pelvic operations have been performed for symptoms produced by a renal or ureteric stone or for pain of a distended renal pelvis from a kinked ureter of a kidney which has undergone ptosis, or over an aberrant vessel speaks for itself. Again, owing to the unusual site, character and radiation of the pain of acute colic, gall stones may occasionally be indistinguishable clinically from renal stone.

To confirm or exclude the origin of an abdominal tumour in a kidney a pyelogram is often necessary. Finally, it is the only certain means of proving the existence of certain kidney lesions, when indefinite symptoms suggest their possibility, for example, an early renal neoplasm, a Gravitz tumour or a

[1] Read at a meeting of the South Australian Branch of the British Medical Association on November 28, 1929.

papilloma of the pelvis of which the only sign may be hæmaturia, hydronephrosis from any cause, as an aberrant vessel, congenital abnormalities, as horseshoe kidney or absence of one kidney.

Renal Lithiasis.

Single stones are usually situated in the renal pelvis and are generally accessible by pelviolithotomy which is the method of choice. Braasch states that 40% of stones are multiple and then often occupy calyces and occasionally (15%) have to be removed through the substance of the kidney. Also large branched stones often have to be dealt with by a combination of the methods.

Recurrent Stone.

By the persistence of the causal agencies after the removal of stones, for example, preexisting foci of infection or other unknown factors, it is possible for stones to reform and there are stone-forming kidneys in which calculi continue to form after all demonstrable foci have been eliminated.

Hager and Magath, of the Mayo Clinic, have found that the *Bacillus proteus* is the organism responsible in several cases of recurrent stone of the bladder and it has been isolated by them in some cases of recurrent kidney stone; they regard the finding of this organism of bad prognosis for reformation of stones.

In addition to this true reformation of stones, it is generally recognized that many recurrences have really been due to the incomplete removal of small stones or little particles which form a nucleus for subsequent stones. To overcome this, in cases of multiple or branching stone, the Mayo Clinic workers use a method of examining the exposed kidney during the operation with a mobile X ray plant in the theatre and a fluoroscopic screen to which is attached a hood going over the radiologist's head. He points out the site of any remaining opacities with a sterile rod. Quinby, of Boston, and Beer, in New York, I saw use little pliable film containers which are placed behind the exposed kidney; a film is taken and rapidly developed.

Another point of technique used extensively was to wash out the pelvis of the kidney with saline solution by syringing it through a rubber catheter passed into the pelvis, to remove any small particles of grit, blood clot *et cetera*.

Ureteric Stone.

Many small stones can pass through the ureter unaided, accompanied by one or more attacks of colic. When a stone becomes impacted in the ureter, it usually does so in the lower portion from the pelvic brim to the bladder. Though many surgeons have their own complicated special instruments for dislodging these, the most common device I saw used was to pass one or more ureteric catheters or bougies up to the stone (one past it if possible) through an operating cystoscope and to leave them in place for twenty-four hours. A little olive oil or glycerin is injected through one and the catheters are withdrawn; during the following days the stone is often passed naturally. Only those that resist such attempts at dislodgement or are obviously too large to pass, are removed by surgical means. Those impacted actually in the intramural portion of the ureter can sometimes be dislodged by being grasped with an instrument through the cystoscope or the meatus may be enlarged by bougies or with special little scissors or by fulguration through the cystoscope. Failing these more conservative attempts they are removed best by opening the bladder and removing them from that aspect.

Although manipulative methods are not always successful, many fewer operations are being performed for ureteric stone than were performed a few years ago.

Pyelitis.

In the treatment of the common condition of pyelitis in the acute stage, if alkalinization of the urine by large doses of citrate and bicarbonate of soda does not control the fever and symptoms, the members of the Vienna school give five cubic centimetres of "Cylotropine" intravenously. If this has no effect, one or two small doses of "Neosalvarsan" are tried.

In the usual infection, after the temperature has been normal for a day or two on alkalinization, the urine is made acid with ammonium chloride 1·8 to 2·7 grammes (thirty to forty-five grains) *per diem* in capsules and hexamine is given in doses of 0·6 to 0·9 gramme (ten to fifteen grains) every six hours, the process being repeated if required. When all signs and symptoms have been absent for a week, the hexamine should be discontinued and a catheter specimen of urine taken for culture before the patient is regarded as cured. Kidd states that in about 40% of cases the infection continues as chronic after the acute attack has subsided and is often unrecognized until the patients have a relapse.

If a condition becomes chronic, it should be investigated and stone and hydronephrosis excluded by X ray examination and pyelogram. Foci of infection must be carefully searched for and eradicated. Hexamine and other medicinal treatment may be first administered for a short time and bladder lavage used, if cystitis is pronounced, but if this does not render the urine sterile on culture in, say, six weeks, a renal lavage offers the most satisfactory results. A fairly large ureteric catheter is passed and the pelvis of the kidney washed out with an antiseptic, one in a thousand silver nitrate solution, a colloidal silver preparation or "Acriflavine" being used. The use of vaccines is generally disappointing. In acute pyelitis of pregnancy which does not react to alkalinization alone, an indwelling ureteric catheter on the affected side for twenty-four hours may be used when it can be borne, with the idea of draining the infected urine retained in the pelvis by pressure of the uterus on the ureter. The temperature usually drops in response and if it rises again, the catheter is reinserted after as long an interval as possible.

Hypertrophy of the Prostate.

Great stress is laid on the preoperative management of all patients with hypertrophy of the prostate

to restore to their best the cardio-vascular and renal efficiency. Hunt states that his mortality rate of patients in good physical condition and with uninfected urine was practically as high as in those of poor condition and renal inefficiency who had had a careful preliminary treatment, so that for the last three years all patients have been given at least ten days' preliminary drainage of the bladder by indwelling urethral catheter. A certain amount of mild infection is thereby produced which acts as a vaccination and increases the resistance to the inevitable infection which occurs in the bladder after all prostatectomies. When the renal efficiency is low, drainage is continued until restored to a point of safety. The indwelling urethral catheter method of drainage is used in this and many other clinics, so that the suprapubic field is left unscarred, facilitating an open, visual, suprapubic prostatectomy to be done, with trimming up of the mucous membrane edge and control of bleeding points. A blind enucleation is seldom seen now except as a second stage after primary suprapubic cystotomy. Suprapubic drainage is used when there are associated bladder lesions, for example, stone or diverticulum, pronounced cystitis or when the renal inefficiency is pronounced, requiring a long period of drainage or in those patients intolerant to the indwelling catheter (about 6%). I must mention here prostatectomy by the perineal route, without stopping to discuss its relative merits. The chief exponents of this method whom I saw, were Young, Hinman and Lowsley in America and Wildbolz in Berne.

Another point emphasized is that the type of anæsthetic used has had a definite influence on post-operative complications and mortality. Ether is rarely seen used, as, though perfectly safe in administration, it has been found more frequently to give subsequent renal depression and pulmonary complications, the two main bugbears of prostatectomy. Gas or ethylene with oxygen is very satisfactory, but has to be skilfully given to give enough relaxation.

Regional anæsthesia was distinctly the most commonly used. For the suprapubic operation Hunt at the Mayo Clinic is using caudal injection with blocking of all the sacral nerves through the posterior foramina and field block of the suprapubic incision, von Lichtenberg, of Berlin, and von Illyés, of Budapest, use caudal injection with field block of the abdominal wall and deep injection into the periprostatic tissues on either side from above behind the pubic bone. For the perineal operation caudal injection alone (Young) or reinforced by lower sacral anæsthesia suffices.

Workers at some clinics in America, at Saint Peter's in London and Marion in Paris were using spinal anæsthesia.

Another advantage of the use of local anæsthesia is that it enables fluids to be administered in quantity immediately after operation. The Mayo Clinic surgeons insist on a daily intake of two and a half to three litres by mouth or supplemented *per rectum* or intravenously.

Carcinoma of the Prostate.

Complete surgical excision of the prostate from the perineum, as described by Young, is only possible and justified in a very small proportion of carcinomata. The next line of attack is radium and this, with a suitable method of application, offers in many cases a definite prolongation of life and alleviation of distressing symptoms and in some cases a clinical cure. Young's method of applying it by the rectum and urethra in a special holder has been given up by most workers, as the mucous membrane is not able to stand a dose large enough to affect the prostate sufficiently without risking a burn. The application of needles set on a trocar and inserted from the perineum with a guiding finger in the rectum, brings the dose into the growth, but it is difficult to get the whole properly irradiated. With needles implanted from above through a suprapubic cystotomy it is again difficult to get enough distribution of dose and often severe cystitis follows and pyelonephritis.

The method used by Gaudy, of Brussels, is to insert the needles directly into the prostate and extensions of the growth, having first exposed it from the perineum in the same way as for perineal prostatectomy. A suprapubic cystotomy is done for drainage, to explore the extent of the growth above and later to keep the bladder at rest, to avoid the severe vesical tenesmus which may otherwise occur, and to lessen the chance of needles being displaced.

Radium and Tumours of the Bladder.

Haphazard technique had placed discredit on the value of radium in bladder cancer, but with improved technique of screening and dosage it has been regaining a place in its treatment, though surgical excision, where reasonably possible, is still the more generally approved mode of attack.

Cystitis is always present with advanced tumours and the results of radium treatment are not so satisfactory when this is severe. The presence of the needles increases the infection and risk of ascending pyelonephritis, so bilateral kidney infection is a contraindication. But even in extensive and inoperable growths it is possible to stop the hæmaturia and cause the tumour to shrink, even to apparent disappearance. The application of seeds through a cystoscope is generally unsatisfactory. A better view and satisfaction that the whole growth is irradiated are secured by small needles or radon seeds inserted through a suprapubic opening, the threads being brought out through a drainage tube.

Fulguration of the main mass of growth, followed by implantation of radon seeds, is practised by Keyes and Barringer for tumours unsuitable for resection. Barringer also has some very good results in the papillary type of carcinoma treated by radium, patients being alive and free from recurrence from five to eleven years after treatment. For papilloma, multiple or single, fulguration by diathermy through the cystoscope is most generally and successfully used, followed by repeated cystoscopic examinations to control a recurrence.

Radium in Epithelioma of the Penis.

Epithelioma of the penis is a type of cancer which reacts favourably to radium and gives the advantage of dispensing with a mutilating operation. A preliminary cystotomy should be done to divert the urine through an indwelling de Pezzer tube and the growth needled. The glands in the groin are most satisfactorily dealt with by surgical excision or if the condition is unsuitable for this, they may be irradiated by radium distributed on a Columbia paste mould.

PREOPERATIVE AND POSTOPERATIVE MANAGEMENT IN UROLOGY.[1]

By W. JOHN CLOSE, M.S., F.R.C.S. (Edinburgh), *Adelaide.*

MY subject is not such a hackneyed one, I hope, as at first sight it would appear, since I expect to interest you in certain fresh aspects of the problems encountered and to that end intend to speed over text book material as fast as possible. Reluctantly enough I shall leave a gap in the scheme of treatment which has been filled by Mr. Jose in his operative procedures, confining myself to the equally important part of the management as carried out in the large urological clinics of the other side of the world.

The first investigation is a purely clinical one and indeed a good clinician should in the majority of cases be able to decide which are the unfavourable signs. As students we marvelled at what we were pleased to term the intuition of our mentors, but soon learnt that what we gave credit to blind intuition was no more than a rapid correlation of facts observed at the bedside and interpreted in the light of vast experience. With profound respect for those present who supervised our early training, I should like to pay tribute to our one-time master and friend, the late B. Poulton, in his almost unerring capacity for making diagnoses and prognoses, without much aid at that time from the laboratory.

To enumerate briefly the procedure necessary in every case:

A careful history of course should be taken and among other things gastro-intestinal symptoms should be given special significance. Quite a number of prostates have been suspected as carcinomata of the gastro-intestinal tract on account of latent uræmic signs referred to this system.

The general condition of the patient, tongue, eyes, breath, colour and respiration should be noted. These are important guides to the renal efficiency.

Cardio-vascular efficiency—heart, arteries and blood pressure—should be estimated.

An examination should be made of the retina and discs, an opinion of the ophthalmologist being sought if necessary.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on November 28, 1929.

The usual office routine examination of the urine must be made. Probably the most important point is the specific gravity and the difference between diurnal and nocturnal specimens must be specially noted. Care, of course, must be taken in collecting a specimen not to deflate rapidly a chronically distended bladder.

Palpation and percussion of all the genito-urinary organs must be carried out. In this item I should strongly recommend bimanual palpation of the empty bladder and the prostate in the supine position, when the patient is not too adipose or muscular. Young's method of palpating *per rectum* on the cystoscope in position gives valuable aid in the diagnosis of prostatic carcinoma and median lobe obstruction.

Instrumental examination should take place in every case unless there are some definite contra-indications, as shown by the examination to date.

A soft rubber catheter or if necessary a stiff one should be passed. If the latter, a large number of different sizes of olive headed, Coudé and bi-Coudé catheters, as well as bougies from filiform size upwards, should be ready sterilized in case of necessity. We thus find the presence of obstruction to emptying and the amount of residual urine.

Cystoscopy.

In regard to cystoscopy, it is surprising how many neglect this aid to diagnosis, prognosis and treatment. Nobody needs to think twice about requesting the ophthalmologist to look at the eye-grounds of a patient of his, so why should the urologist be ignored? The interpretations of the bladder grounds are probably just as abstruse.

In performing cystoscopy and posterior urethroscopy, I generally employ epidural analgesia. We use atropine in the eye to relax the *sphincter pupillæ,* cocaine on the larynx to abolish muscular reflexes, so for the sake of human kindness and the ease of examination we should relieve the distress of cystoscopy, thereby rendering at the same time both the patient and ourselves a service. In the male most of the discomfort is due to the straightening out of the posterior urethra and muscular spasm. Injections of "Novocain" or cocaine into the urethra are almost futile in countering these effects, but if the nerves themselves are blocked temporarily, we can obtain complete muscular relaxation and analgesia. The amount of muscular relaxation attained can be elicited by examining the *sphincter ani* and it is a good guide to the degree of anæsthesia to be expected.

To secure a thorough analgesia the needle should reach as high as the second sacral foraminæ, since the nerve supply of the urethra comes from the second, third and fourth sacral nerves. Success is not invariable in this minor operation, however, as it is not always possible to push the needle higher than the third sacral vertebra without striking cerebro-spinal fluid, thus failing to anæsthetize the second sacral nerve and frequently on account of anatomical peculiarities. Further, it is not a good plan to offend the periosteum unnecessarily in hunt-

ing for the hiatus, else we risk the defeat of our object in relieving pain. In women, unless the bladder is very irritable, it is unnecessary, nor is it worth while in anterior urethroscopy. It is surprising how little affection it causes in the muscles of the legs which depend on sacral innervation. I have not seen one who could not mount and dismount from the table easily and proceed home afterwards without any anxiety on my part for his safety in negotiating the city traffic.

Care must be taken to empty the bladder at the end of examination, as it is common enough for a few hours for retention to supervene. Strangely enough I have seen patients with nervous irritability of the bladder surprisingly benefited by this procedure, though nothing may have been done otherwise for their relief. Of course, one cannot entirely rule out suggestion in such a case and indeed many urologists believe that the Cathelin treatment for enuresis, namely, by epidural saline solution injections, depends for the benefit it often shows on suggestion. Many operative procedures can be carried out by the aid of such an injection: perineal prostatectomy, for instance, though transsacral anæthesia would appear to be the method of choice here, but for the opening of a prostatic abscess or a punch operation it is certainly indicated. I have here a diathermy punch of Kenneth Walker, which you will see is different from other instruments of the same kind in that the projection is coagulated first by diathermy, the fenestra of the sheath itself being the intraurethral electrode. "Bakelite" is the material used to insulate the outer surface of the sheath. The coagulation is performed under vision and a core removed afterwards. Hæmorrhage is negligible and the amount of tissue destroyed is of course greater than that removed. I have used this instrument under epidural anæsthesia without causing any discomfort.

Tests of Kidney Function.

I realize I am treading on dangerous ground in discussing tests of kidney function and must step circumspectly in the squares lest the biochemical bears lying in wait should be only too ready to pounce on me. Especially so since Professor Hugh Maclean has so recently expounded on the subject. I shall therefore limit myself to the tests on which I pin my faith.

The Indigo-Carmine Test.

Indigo-carmine has its value in cystoscopic examination. The dye after intravenous injection is excreted normally in two and a half to five minutes and after this a lowered function is indicated. The surgeon is informed which kidney is the offender or the worse of the two and he may be fairly certain that in the presence of tuberculous kidney, if the good one gives a normal indigo-carmine excretion, it is safe to remove the bad one. On the other hand, the physician is informed when such a comparatively harmless condition as a *Bacillus coli communis* pyelitis has so far affected the renal parenchyma as to make medical treatment ineffective.

To investigate further the function of one kidney, catheterization of the ureters can be performed to determine the urea content of each specimen, as well as for microscopical and cultural purposes.

Naturally, if the result of the indigo test is definite, estimation of the urea content may be unnecessary, particularly if the combined concentration of the kidneys has first been found to be satisfactory.

There are fallacies also in the estimation of function from the urea content, as diuresis may occur if fluid has been drunk less than two hours before or through the inhibitory effect on the tubules caused by the mere presence of the catheters.

Concentration Tests.

Concentration tests are numerous and include those in which the dyes such as phenol-sulphonephthalein are used, but I shall not attempt to allude to them all.

In Maclean's test attention is paid to the urea concentration and it is on the whole the most valuable; with others attention is paid to the concentration of total solids, the so-called dilution tests, a simple one of which I shall describe. I have alluded above to the comparison of the specific gravity of day and night specimens, which is virtually all this test comprises. The drinking of a considerable quantity of water, say nine hundred cubic centimetres (thirty ounces) in the morning should give a maximum (night specimen) and a minimum (day specimen) concentration of solids. The urine of the first few morning hours, nothing having been drunk over night, is the first and that collected during the second hour after taking the nine hundred cubic centimetres of fluid, the second specimen. When the bladder is being drained in hospital, a nurse can do this every day or second day and chart the result.

A patient with a urological condition such as an enlarged prostate should manifest a difference of ten points, for example, 1015 to 1005. The patient with an azotæmic kidney may show only a difference of one or two points, the specific gravity remaining low in both specimens; the hydræmic kidney patient may show a similar small difference, but on the other hand the specific gravity is high in both. A fairly similar result may be obtained in failing circulation to that found in hydræmic kidney, on account of the retention of fluids.

Blum, of Vienna, advises a record of the weight of the patient together with accurate estimation of the quantities passed, in order to detect the retention of fluids more easily.

Calvert's test[1] combines urea concentration and dilution. It is used by Mr. Swift Joly, at St. Peter's Hospital for Stone, as a routine for all his patients with prostatic conditions, but as it is obviously not an office procedure, I shall not discuss it further.

The Blood Urea Content.

Diseased kidneys may not cause a high resting blood urea, but are unable to cause concentration. As Sir John Thompson Walker says, the blood urea concentration figure tells you when to withhold your eager hand, but does not tell you when to go ahead. The blood urea curve, however, is a different story. Archer, of Saint Bartholomew's, has elaborated this procedure which he names the urea tolerance test, and it is conducted in much the same way as the sugar tolerance test.[2] [3] I have made casual reference to this test in a recent paper and consider that it merits further comment.[4]

He uses a very convenient micro-method which has proved a boon where frequent estimations on the same patient are desirable and in children it is invaluable.

Urea differs from sugar in that it is present only to be excreted, yet in spite of this the blood level is usually between 0·02% and 0·04%, urea being what Cushny terms a medium threshold substance. If say fifteen grammes of urea are ingested, the blood urea temporarily rises from the resting twenty to forty milligrammes to another five to ten milligrammes in thirty to sixty minutes. The return to the previous state is complete in one hundred and twenty minutes.

A deficient tolerance may be indicated by one or more of the following: Resting blood urea above normal, rise above fifteen points, recovery not complete within four hours (allowing five to six milligrammes hourly fluctuation). Any of the indications mentioned should cause one to postpone operation or to perform a two stage operation. In regard to elderly people of the prostate age, further research seems to be necessary to determine the normal and the risk level, as no doubt the curve is lengthened in later life, just as the sugar curve may be.

I have made use of the curve in prostatic conditions when other tests were not convenient, for example, the bladder was being drained and continuously flushed to combat sepsis or intense hydrotherapy was being pushed. So far I place most reliance on the length of the curve; if it has not reached the resting level in four hours, it is decidedly poor.

The micro method is briefly as follows. Only 0·2 cubic centimetre of blood is required and this can be obtained by pricking the finger and squeezing if necessary, for example, in a child. It is incubated and precipitated afterwards, being finally Nesslerized and compared colorimetrically with a standard solution of sulphate of ammonia.

Urease has previously been difficult to obtain pure, the soya bean flour commonly used containing impurities such as ammonia. Tablets made by Dunnings are obtainable through the British drug houses, though I was surprised at the difficulty of getting any in Australia, which seemed to indicate that they were not very generally used here. I have now been able to obtain a supply from England and through the courtesy of Dr. Bull and the energy of Mr. Cowan, of the Adelaide Hospital laboratory, this micro method has been tried along with other methods in a series of cases and the latter now informs me that he has willingly discarded the old for Archer's method which is proving so satisfactory.

The advantages of such a method must be very obvious. Children seem to mind the proceeding very little and at Great Ormond Street and the East London Children's Hospital it was not uncommon to see it done about every other day on the same subject.

I was very interested to see the gradual rise of blood urea through bowel absorption soon after operation and its gradual fall to safe levels in some patients of Mr. Tyrrell Gray who was attempting the transplantation of the ureters into the ileum in *ectopia vesicæ*.

Tests for General Use.

I have occasionally been asked by country practitioners who have not the handy assistance of a laboratory as to the most convenient tests for them to use. My reply is to recommend first the urea concentration test of Maclean. This can be done very simply in the office with a Hind's modification of a Doremus's ureometer. For clinical purposes it is quite accurate enough. Bromine fumes in the consulting room are a drawback, but it is possible to obtain the bromine in capsule form, so that it can be broken in a stoppered bottle in contact with the caustic soda solution and no fumes escape. Secondly I recommend the dilution test as described above and thirdly salivary urea can be substituted for the blood urea for practical purposes. I find that it roughly parallels the blood urea findings. For its technique I must in such a short paper refer you to an article by Hench and Maldich in the *Proceedings of the Mayo Clinic*, 1923.

The Distended Bladder.

I have made respectful reference to the emptying of an extremely distended bladder. Most of us recognize the necessity of doing this gradually and this does not apply only to elderly men with prostates, but to patients with stricture and the children suffering from congenital obstruction. Some men seem to think that the risk applies only to the first series, but even in the others the kidney has been so hampered by back pressure that the engorgement subsequent on rapid emptying may be very serious.

Supposing in your practice you are called out to a good patient "in the very witching time of night" for the purpose of relieving the result of the day's indiscretions, it naturally appears advisable for the convenience of both of you to get it over quickly and be thankful that there is no afterbirth to wait for. Actually the best plan on finding the bladder well above the pubis is to leave in a ureteric catheter about number 7 or a filiform bougie if this can be inserted. The emptying will then be very slow. If unsuccessful, a rubber or bi-Coudé gum elastic catheter may penetrate, not more than three hundred cubic centimetres (ten ounces) of urine

being then withdrawn, the catheter clipped and instructions left for a similar amount to be removed every four hours. I have used a modified Young-Shaw apparatus in hospital for the same purpose, finding that it gives a very gradual decompression and at the same time obviates the admission of sepsis.[5] Its adoption in spinal injuries before the onset of reflex micturition takes place is also recommended by Young, to avoid the cystitis which is unavoidable by intermittent catheterization.

Radiography.

Routine pictures of the whole urinary tract are first taken and at the same time as the ureteral catheterization is carried out a pyelogram is made when indicated. I make use of Braasch's technique as performed at the Mayo Clinic.[6] A Braasch-Bumpus table, with Potter-Bucky diaphragm incorporated, can be lowered or raised to the Trendelenburg or reversed Trendelenburg position and is a great comfort. The latter position is especially necessary for viewing the ureter, the catheter being almost withdrawn.

Cystography has an application where the use of the cystoscope is limited, for example, in diverticula of the bladder.

Pyelography in Children.

A scheme for pyelography in children has been worked out by Addison, of Great Ormond Street, since the operation must necessarily be carried out under anæsthesia, when the signs of pelvic distension will not be available, as in the adult.

He found in cadavers that the capacity of the child's renal pelvis commenced at about one cubic centimetre for infants of one year, increasing by almost one cubic centimetre for each year up to five years.

His double catheterizing cystoscope of 14·5 *Charrière* scale I have already shown you here. It fulfils all his requirements and is entirely boilable, telescope, sheath and cable.

Preparation for Operation.

The investigation being now complete and the question of operation decided, I shall trespass just long enough in the hospital wards to see my patients fully prepared for the last stages of the cure.

Preliminary drainage is necessary in a variety of conditions which it would be difficult to enumerate here, but the following indications should guide one:

1. To improve the function of patients with obstruction due to enlarged prostates in particular. If there is any doubt about the renal function, carry out preliminary drainage in all such cases. Marion, of the Lariboisière, Paris, performs suprapubic cystostomy in all men over fifty-five years and these naturally comprise the majority of his patients with enlarged prostates. The inlying catheter is frequently used, but suprapubic drainage is preferable. A large drainage tube through a bladder incision gives best results, but it is often a better way to insert a large Morson's trocar and cannula, passing a de Pezzer catheter number 28 *Charrière* scale through the cannula, local anæsthesia being sufficient in patients who will obviously have to wait some time before further operation. In this way the patient can get about for at least nine to twelve months and with precautions prevent gross sepsis of the bladder. It is a convenient way also of performing gradual decompression when the inlying catheter is not well tolerated.

2. To combat gross sepsis. Thomson Walker employs an inlying catheter, connected with a drip of one in 15,000 silver nitrate solution, the exit being through a suprapubic tube and cap. The same method is used by him for postoperative prostatic hæmorrhage, a rare occurrence after his open operation, but always to be looked for. The Young-Shaw apparatus is also sometimes useful in cystitis, though it is more useful as a prophylactic. A similar apparatus, though in my opinion not so convenient, was recently designed by Cuthbert Dukes, pathologist of Saint Mark's, to prevent the inevitable urinary infection that occurs after excision of the rectum.

3. As a preliminary to most plastic operations on the urethra. One of the exceptions is a new operation for hypospadias by Edmunds, of Saint Thomas's, performed in three stages. I have seen excellent results from it, but am of the opinion that a good part of the credit should be given to Hamilton Russell whose operation it closely resembles. This is my excuse for mentioning it here. A ruptured urethra causing retention should always be treated primarily by cystostomy.

Whilst on the subject of the urethra, I would ask your forbearance with a further digression. I was agreeably surprised with the dexterity of the French surgeons in their urethral work and the number of ruptured urethræ amazed me. The explanation was forthcoming when I witnessed the tail end of a street brawl one evening. It was quite evident that the Frenchman realizes that the most vulnerable parts of the male anatomy are the face and perineum and the most effective *vis a tergo* is not the British weapon, the fist, when one's victim lies at one's feet. The police officer who summarily settled the matter was also fully aware of the efficacy of attack from the same quarter and with the same weapon.

Methods of Combating Nitrogenous Retention.

Apart from drainage, the only means of any use for combating nitrogenous retention are hydrotherapeutic. Five to nine litres (eight to fifteen pints) should be aimed at per day, the amount depending on the height of the resting blood urea and as much as twelve litres (twenty pints) may be administered in the worst cases. Such an amount of course is a big strain on the circulation and especially in patients with high blood pressure must be watched from this aspect. The heart will probably need some assistance at the same time by digitalis and the cardiologist is a most useful man to consult during such intense hydrotherapy.

After-Management.

It is not in my province to discuss in detail the immediate treatment of patients operated on, as my scheme is to take up treatment again after their return from the wards. I shall content myself then merely with an outline of the work of a large urological out-patient clinic.

The various functions of such a clinic comprise routine investigation of patients much as I have detailed it above, with variations of course as to technique and favoured tests. Treatment is conducted for strictures, intermittent dilatation being the method of choice in most cases and the stricture department forms far and away the biggest part of the clinic, the follow-up treatment of internal urethrotomy patients and the now rare external urethrotomies, as well as periodic examinations of patients subjected to plastic operations. Whether the operation has been one of internal or external urethrotomy or where no operation has been performed at all, dilatation must be continued at intervals for years and sometimes for a lifetime.

At some clinics surgeons content themselves by passing bougies up to the limits of the meatus, numbers 14 to 16 English or 26 French, continuing these at long intervals for the patient's lifetime. Others believe in tiring out the stricture by means of a Kollman's dilator; 30 to 35 *Charrière* scale can be attained in this way, the limits of dilatability of the urethra being watched by the air distension urethroscope. It is said that many strictures thus treated do not contract again.

Prostatectomy patients are followed up periodically and endovesical treatment by diathermy is given to most of those who have trouble later from ledges, small adenomata that have escaped removal on account of their insignificance at operation or stricture formation in the posterior part of the urethra through contraction of scar tissue.

Cystitis which so frequently persists after prostatectomy, can be watched and regulated; it is a disappointment to find a good operation robbed of its benefits by an intractable cystitis.

Patients with papillomata of any size are usually first treated under anæsthesia and as in-patients. About three weeks later treatment is continued in the out-patient department without general anæsthesia, by diathermy and it is even necessary to keep patients apparently cured under observation at intervals of three months for the first year and six months for the second. Small seeds can be detected then and treated through the cystoscope at once. Even at the neck of the bladder small papillomata and adenomata can be treated effectively in the out-patient department. A retrograde instrument of Swift Joly allows for this. The lever is a departure from the usual Albarran type, the operating attachment takes a number 11 instrument comfortably and the parts are boilable. I have it on view here.

Mr. Swift Joly's special genius is a mechanical one; his air distension urethroscope is known to some of you and I have already shown you his apparatus for sterilizing ureteric catheters, which allows for their introduction by avoiding contact with everything but the cystoscope itself.

When tuberculosis has been treated by nephrectomy and the disease still persists in the bladder, the patients are followed up and given necessary treatment in such a clinic. "Gomerol," a guaiacol preparation, seems to be in general favour on the Continent and in England for this condition. Although it is a French preparation, the Germans are equally enthusiastic about it.

One must not forget the patients with cancer and tuberculosis that come up for relief only, nor the many patients with functional urinary disorders, nor the cystitis that follows other than urological operations. Obviously a general surgical clinic is not the place for any of these, if we mean to give them the considerate treatment which all the surgical world considers their due.

Conclusion.

I hope with others of you who are interested in the subject, that Adelaide will not have long to wait for the establishment of a urological clinic as well as a number of other special clinics which her dignity and reputation as a teaching centre demand.

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Reports of Cases.

POISONING BY ARSENATE OF LEAD.

By ARTHUR PALMER, M.B., M.S. (Edin.), F.R.C.S. (Edin.), *Government Medical Officer, New South Wales*,

AND

STRATFORD SHELDON, M.B., Ch.M. (Sydney).

ALTHOUGH arsenate of lead is so commonly used in the orchard and garden for the killing of various pests, we have been unable to find a record of death from the taking of this substance.

Nicotine is another poison available in much the same way and, although providing a quick exit from this world, is rarely used.

In this country the poisons most frequently used by the suicide appear to be carbon monoxide, cyanide of potassium, strychnine and lysol and its relatives.

The following two deaths from the taking of arsenate of lead are here recorded.

A Chinese, aged thirty-four years, died on July 19, 1928, after a long illness, death being certified as due to duodenal ulcer and myocardial degeneration. On August 20 of the same year the body was exhumed and subjected to analysis. It was considered that the body was unusually well preserved for the length of time that had elapsed since death. In 2,721 grammes (six pounds) of the viscera, chiefly liver, 0·7 grain of arsenic and 1·5 grains of lead were found; in 227 grammes (half a pound) of bone three milligrammes of arsenic; and in 6,249 grammes (fourteen pounds) of muscle two grains of arsenic, while traces of that metal were also found in the toe nails. The wife and her lover who married very soon after the death of the Chinese, were tried for murder and acquitted.

A woman, aged fifty-five years, was admitted to a mental hospital in February, 1927, suffering from acute depression with delusions and suicidal tendencies, but absconded in August, 1928. On the morning of September 2 of that year she was seen to have some white powder on her lips. On being questioned she told one person that it was arrowroot, another that it was tooth powder. On this day she did not seem to be ill, but next day remained in bed, vomiting frequently. At 2 p.m. she was seen by a doctor who prescribed a sedative mixture. She was restless during the night and next day, September 4, for the first time complained of pain in the abdomen and continued to vomit everything given her. It was only then that she confessed that the arrowroot had really been arsenate of lead which was kept in the house. She was removed to hospital and died at 7 a.m. on September 5. Analysis of the viscera showed the presence of arsenic and lead in considerable quantity, while quite a large quantity of arsenate of lead was still seen to be adhering to the rugæ of the stomach. At the autopsy there appeared to be little irritation of the stomach, but considerable irritation of the small gut.

We are indebted to Mr. Doherty, the Government Analyst, for the following note on the toxicology of lead.

The Toxicological Aspect of Arsenate of Lead.

The felonious use of arsenate of lead, the compound so largely used in the spraying of fruits and vegetables, invites an investigation as to its potency in the destruction of life. Men have died after its ingestion and the insertion of it into auger holes in tree trunks has been followed by the death of the trees. This later action, by the way, shows the value of experiments concerning the decomposition of the compound by the action of water (see "Decomposition of Lead Hydrogen Arsenate by Water," by McDonnell and Graham, *Journal of the American Chemical Society*, 1917, page 1912). These experiments are not concerned with the possibility of the occurrence of free arsenic acid which, though prohibited, may be present in some crude specimens of the arsenate to quite a notable degree.

Arsenate of lead is known variously as ordinary lead arsenate, acid lead arsenate, diplumbic hydrogen arsenate, dilead arsenate.

An analysis of a commercial sample gave the following result: arsenic acid (As_2O_5), 33·0%; plumbic oxide (PbO), 63·0%. This does not quite agree with the formula generally allotted to the compound: $PbHAsO_4 H_2O$.

Arsenate of lead is largely used as a pest eradicator in orchards and vegetable gardens and it owes its popularity to the fact that, though it is practically insoluble in water and thus not injurious to the vegetal surfaces upon which it is sprayed, it is poisonous when ingested by animal life generally. Thus are insects destroyed and no doubt lower forms of life. That is to say, that sufficient decomposition takes place and sufficient arsenic is dissolved by the digestive system to become poisonous to the host. It is thus from one point of view an ideal product, provided not too much remains on the crop. It destroys the parasite without injury to the plant. But although lead arsenate is so insoluble in water, it is slowly decomposed by contact with that liquid and arsenic acid is formed. The concentration of the arsenic acid in the water is so low, however, that unless the water be constantly renewed, the decomposition with small amounts of water is inappreciable. If the water be constantly renewed, in the course of time the lead arsenate is decomposed, with the formation of a basic salt. The reaction which is reversible, is thus represented:

$$5PbHAsO_4 H_2O \leftrightharpoons Pb_4(PbOH)(AsO_4)_3 + 2H_3AsO_4.$$

In the transformation of lead arsenate to the basic arsenate 40% of the total arsenic present should be removed. In an experiment in a Soxhlet apparatus, in which hot water was used continuously for nine months, 38·5% of the arsenic was removed, which is a close approximation to the theoretical amount. In another experiment two grammes of lead arsenate lost 0·0438 gramme in one month and again where the lead arsenate was subjected to the action of constantly changing water for five months, 6·5% of its arsenic was dissolved. These two last-mentioned experiments were with cold distilled water.

Reviews.

EPIDEMIC ENCEPHALITIS.

THE literature dealing with the various diseases of an epidemic nature has reached enormous proportions. So scattered and so varied are the references that the investigator of any one of these diseases, who would consult the literature fully before mapping out his programme, would find little time left for any original work. Though epidemic encephalitis is a comparatively new disease, having been described about ten years ago, the survey on its ætiology, epidemiology and treatment made by Dr. Josephine B. Neal and Dr. Helen Harrington for the Matheson Commission under the chairmanship of Dr. William Darrach, contains a bibliography of 429 pages and no less than 172 references to authors whose names begin with A.[1]

When it was represented to Dr. William John Matheson how much work was being done throughout the world on epidemic encephalitis, he realized the advantage of having the knowledge thus obtained collected and epitomized, so that it might be readily available for consultation. To carry out this idea, he appointed a commission and generously financed the undertaking. The work before us, published at the nominal price of three dollars, which is less than the cost of printing, is the result of his generosity. In addition Dr. Matheson is financing a two years' study of the value of different methods of treatment of the disease as well as experimental work at Porto Rico and at the Pasteur Institute at Paris.

The survey is divided into sections. In the first, on ætiology, each reference to this aspect of the disease is summarized in a series of epitomes of each author's work without comment or bias. There are thus brought under review many articles dealing with a possible streptococcal causative factor in contrast with the still more imposing array of works indicating that the responsible agent is probably a filter-passing virus. All the varied and sometimes conflicting experimental results find a place.

In the second chapter, on other types of encephalitis that following vaccination, is fully considered. The chapter on treatment which follows, reveals that there is as yet no method which can do more than alleviate occasional symptoms. In the chapter on epidemiology there is much of interest. Under the heading of Australia there is a summary of Australian work on the so-called *X* disease, so fatal in 1917 and 1918 in certain parts. The similarity of the Australian disease to one of the forms of epidemic encephalitis in Japan is of much interest.

Workers interested in epidemic encephalitis have in this volume an up-to-date epitome of the literature on the subject. How valuable this is any one who consults its pages, will at once see. Dr. Matheson should be thanked for his generosity in enabling this summary to be compiled

[1] "Epidemic Encephalitis, Etiology, Epidemiology, Treatment: Report of a Survey by the Matheson Commission," William Darrach, Chairman; Joseph B. Neal, Director of Survey; Helen Harrington, Epidemiologist. New York: Columbia University Press; 1929. Pages xiii + 849, with charts and tables. Size 7½ inches × 5 inches. Price $3.00.

and his commission should be congratulated in the successful accomplishment of a very big undertaking. A supplement is promised to appear early in 1930.

BODY-SNATCHING FOR ANATOMICAL STUDY.

IN a book with the startling title, "The Sack-'em-up Men," a title evidently chosen to shock the reader, Dr. J. M. Ball describes the study of anatomy in ancient times, its decay under religious proscription, its revival at the *Renaissance* and its modern progress.

Even today there is something horrible and gruesome about the study of anatomy. What was the state of affairs one hundred years ago we can scarcely conceive. We work in clean and beautifully equipped rooms on well preserved bodies. Our great-grandfathers had only putrid subjects. It is appalling to think that they used bodies which after having been buried for hours or even days, were exhumed, dragged out of their broken coffins, torn from their graves and stolen by "body-snatchers" or "resurrectionists" for sale. We are not told what preservatives were used, but how foul must have been the stench of the dissecting rooms. Small wonder that students often sickened and died of slight wounds acquired at their work.

It is shocking to think that only from the ghouls who, evading the watch of grieving relatives anxious to protect the mortal remains of their loved ones, desecrated graves and stole bodies, could otherwise honourable professors and teachers of anatomy obtain the necessary material for instruction of medical students. What wonder if among the rogues who robbed churchyards, were some who did not shrink even from murder!

Yet enthusiasm for seeking out the secrets of Nature could overcome all repugnance and reputable teachers consciously bought stolen material, nay, sometimes themselves stole, rifling graves with their own hands. Such things are nowadays unknown.

The very handsome, beautifully printed, copiously and magnificently illustrated volume before us paints a vivid and entrancing, if horrifying, picture of the difficulties in which anatomists worked.

The author displays a wide erudition and cannot resist the temptation to dilate at length on many a matter quite unrelated to his subject, such as the character of Frederick II, of Sicily. Sometimes unhappily he lapses into ridiculous errors, as when he tells us that Aristophanes, the dramatist, laboured near the banks of the Nile in the age of Alexander the Great; Aristophanes had, of course, flourished a century earlier in Athens.

Dr. Ball is an American ophthalmologist. He marrs our pleasure in his impressive if somewhat incoherent work which, indeed, suggests a collection of essays rather than a single composition, by indulgence in caustic remarks and frank disapproval of many deplorable events in Scottish and English history.

SLEEP.

A WELCOME addition to the minor monograph series will be found in the volume, "Sleep and the Treatment of Its Disorders," by Dr. R. D. Gillespie.[2] Its value lies not in its originality, but in the bringing together from various sources of a large number of observations which are not found in the average text book.

Sleep is an important topic to the practitioner and specialist in any department, so a wide range of readers should be assured. The contents of the monograph consist of chapters on physiology and patho-physiology, the effects of sleeplessness, pathology of sleep, treatment, theories, a *résumé* of the nature of sleep and a useful list of references.

The author is to be congratulated on his impartial handling of the various psychological and physiological aspects of sleep. The tendency to take sides which is so easy in such a controversial topic, is avoided. The manner in which the section on treatment is handled betokens wide clinical experience. Amongst sundry classes of patients with insomnia attention is drawn to those who show anxious preoccupation. They deny indignantly worry before bedtime and protest that they "think of nothing." As Dr. Gillespie points out:

> This is the kind of person who expects to be able to worry all day and yet to sleep at night simply because he changes the topics of his preoccupation. In point of fact, by thinking of "nothing," by which he means nothing unpleasant, he is making a tremendous effort at repression of his daytime perplexities and it is the effort at repression which keeps him awake.

The art of procuring sleep in children by the methods which include making the preparation for bedtime part of play, will be found helpful to the practitioner.

Dr. Gillespie believes in the use of hypnotics provided four rules are obeyed. They should never be used alone, but with other methods. They should be used only when other methods have failed. Procrastination should be avoided and adequate doses insured. Explanatory reassurances as to drug addiction and where necessary insanity should be given.

The student who is interested in the intricacies of the various theories of sleep will find a well assorted summary of the various modern conceptions. Though Pavlov's work on inhibition receives most mention, the author is forced to state that an "explanation" must take account of data from many fields. Space unfortunately forbids a fuller recapitulation of this interesting question.

The book is well worth a perusal and a place amongst the works of reference.

GYNÆCOLOGICAL METHODS.

"GYNECOLOGIC TECHNIC," by Thomas H. Cherry, of the New York Post-Graduate Medical School, is a book which the student and practitioner will find most useful.[1] It is the work of a keen teacher setting forth his own teaching. It is not a comprehensive text book or book of reference. The writer confines himself to the description of treatment that he uses himself. Many methods commonly set out in gynæcological text books are therefore omitted. As illustrating this, it may be mentioned that trachelorrhaphy, low cervical amputation and Sturmdorf's operation are not described. The author has his own way of excising the cervical endometrium, prefers electrocoagulation to trachelorrhaphy and has no use for low cervical amputations. Other notable omissions are panhysterectomy for cervical carcinoma and the shortening of the round ligaments by the inguinal route.

The book covers a wide range, however. The first four chapters are devoted to the herniæ and to the various plastic operations of gynæcology. The teaching here is on the whole in accord with modern practice. Five chapters are given to intraabdominal operations and one of these is a short account of operations on the intestines. Malignant disease of the rectum is not mentioned. The writer is evidently troubled to decide what should be omitted.

The use of radium and radium emanation in carcinoma of the cervix is described and Regaud's technique and the author's method are set forth. No account of the immediate or ultimate results of radium treatment is given.

Chapters are devoted to anæsthesia including local anæsthesia, post-operative care, diathermy, sterility (wherein gas inflation tests and utero-salpingograms are described), gonorrhœa, uterine hæmorrhage and a few of the urological conditions which the gynæcologist encounters.

[1] "The Sack-'em-up Men: An Account of the Rise and Fall of the Modern Resurrectionists," by James Moores Ball, M.D., LL.D.; 1928. Edinburgh: Oliver and Boyd. Royal 8vo., pp. 247, with illustrations. Price: 16s. net.

[2] "Sleep and the Treatment of its Disorders," by R. D. Gillespie, M.D., M.R.C.P., D.P.M.; 1929. London: Baillière, Tindall and Cox. Crown 8vo., pp. 276. Price: 7s. 6d. net.

[1] "Surgical and Medical Gynecologic Technic," by T. H. Cherry, M.D., F.A.C.S.; 1929. Philadelphia: F. A. Davis Company. Royal 8vo., pp. 700, with illustrations. Price: $8.00 net.

The Medical Journal of Australia

SATURDAY, MARCH 22, 1930.

The Death of Henry William Armit.

It is with the most profound regret that we have to announce the death of Dr. Henry William Armit, Editor of The Medical Journal of Australia. On the morning of Saturday, March 8, 1930, he was in his usual place at the office of the journal. He admitted that he had a sore throat, but with typical stoicism refused to agree that it was more than a trivial affair. That evening he complained of not feeling well and went to bed. A rigor and fever followed. The illness quickly developed and his medical attendants were ere long faced with the dread diagnosis of a streptococcal septicæmia. The infection was so virulent and his resistance was so lowered by constant devotion to his labours, by his refusal to rest and by the mental stress of recent events that those who knew him well, realized that there could be but one ending. The end came swiftly on the evening of March 12.

His death has come with such appalling and tragic suddenness that it is difficult to realize that it has taken place. It is certainly impossible to gauge the extent of the loss to this journal and to the whole profession in Australia.

All men have opportunities of influencing for good or evil those with whom they come into daily contact; their example and precept do not pass unheeded. Medical practitioners have larger opportunities than the majority of other men; they are brought into intimate relationship with the sick and suffering at a time when the minds of the latter are receptive, when force of character can make a lasting impression; they are regarded by those who are not ill, as beings, intensely human, who may be used as a tower of refuge in time of trouble. A medical journalist who loves his profession, has the power and privilege of influencing his *confrères*. His power is the power of the press, supplemented by a scientific training and by an inherent ethical outlook. Armit regarded privilege and power as sacred.

He came to Australia in 1914 peculiarly well prepared to control the destinies of the new journal, The Medical Journal of Australia. He had been through the apprenticeship of general practice. He had known the difficulties and hardships of the general practitioner, he had had experience of the trials and tribulations and he knew something of the triumphs. He had worked laboriously in the laboratory, had lived on the laboratory worker's proverbial pittance, had known what it was to work for months making minute observations which seemed to lead nowhere, and had had the satisfaction of making useful additions to knowledge. He received his training in journalism as a member of the staff of *The British Medical Journal* at the hands of that great master, Dawson Williams. Small wonder was it that he made a forceful impression on the medical mind of Australia.

As Armit had learned, so he taught. He held that a medical journal is a post-graduate teaching medium. He sought to emphasize scientific fact and to distinguish the well-founded observation from that of doubtful worth. An observation made either clinically or in the research laboratory was to him worthless unless it could withstand the most critical investigation. He was a diligent seeker after truth. He held, and rightly, that research deliberately planned towards a preconceived conclusion is unreliable. He sought to prevent disease and most of his journalistic effort was expended in this direction. To determine the cause of disease was to him more important than to discover an empirical cure and in treatment he demanded that the patient be the first, nay the only, consideration. His soul abhorred the self-seeking collector of good statistical results, for he realized that the patient did not receive his due at the hands of such a man. He was most jealous of the honour of his profession. Any impugnment of honour or infringement of ethical code was to him a call to arms, a call to which he responded without regard to personal consequences.

Such were some of his beliefs and some of the ideals which activated him, and to which he was entirely devoted. They may be set up as a standard for emulation and those who follow them will be accounted worthy. At a later date a full account of his career and of his attainments with an appreciation of his personal qualities will be published and those who so desire, will be given an opportunity of paying tribute to his memory.

Current Comment.

ASTHMA.

AN asthmatic person may be in such an excellent state of health that stimuli do not produce asthma; he may be in an unstable state characterized by attacks of asthma, occurring at intervals and occasioned by slight stimuli; he may be subject to repeated attacks of asthma, without any complete freedom from dyspnœa, in other words he may be in the *status asthmaticus*. As far as the attack itself is concerned there is a preparoxysmal stage, followed by the paroxysm and the postparoxysmal stage. In November, 1928, Barber and Oriel reported an important series of biochemical observations on the allergic state and described them as occurring in a cycle. They described a rise in the amino acid content of the blood; a fall in the chloride content, particularly of the corpuscles, regarded as due to the chloride being taken up by the tissues; retention of the chloride until the paroxysms have ceased; a rise in the urinary secretion of ammonia; deposition of urates in the urine in the preparoxysmal and paroxysmal stages; increased intensity of the ether reaction in the urine while active symptoms are present; diuresis with decreasing acidity and sometimes alkalinity in the postparoxysmal stage. They laid down certain principles of treatment based on these observations.

As a result of work carried out at the Asthma Clinic at Guy's Hospital, G. H. Oriel has continued his observations.[1] He states that it has become apparent that asthmatics do not fall into one biochemical group. He has investigated the condition of one hundred and ten patients and has divided them into two contrasting types. The first type was found in 20% of the patients. In this type the first symptom occurs in infancy and is usually manifested in eczema; this is accompanied by or alternates with attacks of asthma, often diagnosed as bronchitis. As the child developes, the eczema becomes less pronounced and is replaced by prurigo. This is often accompanied or replaced by cyclical vomiting and in later life by migraine, but asthma remains the most distressing symptom. In this type there is a low blood sugar content, a positive Van den Bergh reaction occurs and the amino acid content of the blood is raised. The urine is concentrated and urates are deposited. The second type was commoner and comprised 66% of the patients. The immediate precursor of the first attack is stated by Oriel to be frequently an acute infection of the lungs, such as pertussis and bronchitis or pneumonia. The onset is often at puberty or the climacteric. An example of asthma of this type is quoted by Oriel—a man, aged forty years, who had had two attacks a day for twenty years and who had no other allergic manifestations. In this type of asthma Oriel found a low blood sugar content, absence of reaction to the Van den Bergh test, a normal amino acid and a normal bicarbonate content of the blood. The titrable acid of the urine was low, the ammonia combined acid was relatively high and the urea concentration was low; the volume of urine passed was normal. When a patient with this type of asthma was examined while he was in the *status asthmaticus*, it was found that the ammonia content of the urine was high, the urine was alkaline and it contained much diacetic acid. When the patient was free from asthma, these findings were normal.

Of the one hundred and ten patients in the series 56·4% had or had had other allergic manifestations; 16·3% had had eczema, 15·4% migraine, 13·6% cyclic vomiting, 16·3% urticaria and 6% hay fever. As already pointed out Barber and Oriel have discovered a definite similarity in the biochemical changes in these conditions. It was found that all these conditions gave rise to an ether reaction in the urine and this has been shown by Oriel to be due to a complex nitrogenous substance allied to the secondary proteoses. When it is injected into the skin of the patient from whom it was isolated, this substance gives rise to a reactive redness. The substance is found in the urine only during an attack. Oriel points out that it is not histamine, because it causes no contraction when it is added to the perfusing fluid of an isolated guinea-pig uterus preparation. The evidence quoted by Oriel in favour of its specificity is of interest. Although it causes a reaction in the skin of the patient from whom it is obtained, it fails to cause a reaction in normal controls; when injected in minute doses such as one one-hundred thousandth part of a gramme, it causes an acute recrudescence of symptoms; in other allergic conditions tests made from one patient to another failed to yield a reaction; when this substance was isolated from the urine of a patient who was free from attacks, it gave no reaction in his skin, but when he was injected with "pollacin" and an attack of asthma was induced, his urine was found to contain a substance which gave positive reactions in his skin, but not in that of controls.

It must be remembered in the first place that asthma is a manifestation of hypersensitiveness and that other conditions such as urticaria, angioneurotic œdema *et cetera* are different phases of the same phenomenon. Oriel's observations have been confined to the biochemical changes in the blood

[1] *Guy's Hospital Reports*, October, 1929.

and urine. They cover the results of the manifestations of hypersensitiveness in certain patients; they are the shadow, not the substance. The two must not be confused. It is not sufficient to be content with the shadow, but by following it the substance may be discovered. Oriel's results would appear to indicate a different process in the two types of asthma described by him. This difference may or may not exist. In both the reagin, the property of the plasma which gives it the power of reaction, is probably identical. The atopen, the substance capable of exciting the symptoms, is probably different. The atopen may be an agent introduced from without the body; it may be a bacterial toxin or a foreign protein; it may be one of the products of metabolism. Some atopens are recognizable. The reagin has not been determined and thus the essential nature of sensitization is unknown. The changes described by Oriel as occurring in his first type are indicative of hepatic damage. He states that from a biochemical point of view the chief feature is instability: the asthmatic is constantly switching over from a condition of acidosis to alkalosis with corresponding urinary changes. Ketosis is easily produced and sugar is easily exhausted. He regards the problem of treating the diathesis as resolving itself into making the biochemical behaviour of the asthmatic stable. He has attempted to do this by giving glucose and spirits of ammonia. The patient whose history he quotes as exemplifying the good results of this treatment, is one to whom he refers as an example of those suffering from the second type of asthma. It would be supposed that glucose would be more useful in the first type. Oriel endeavours to explain the action of the ammonia. He arrives at no definite conclusion, but points out that giving ammonia by mouth to the type of patient whom he describes, seems to increase the free acid in the urine and diminish the ammonia combined acid, thus restoring the ratio to normal. This treatment may be useful, but it is merely symptomatic. Oriel states as a sort of after thought that "another part of the problem of asthma is the question of sensitization." This is the fundamental part. As already pointed out, Oriel has made observations on only one aspect of the biochemistry of the condition, albeit an important aspect. The presence of histamine-like bodies and the reason for the appearance in the urine of the substance described by him must be studied. It is not sufficient to treat the patient according to certain clinical biochemical findings. The way in which those findings are occasioned, must be determined. Asthma must be a problem in preventive rather than in curative medicine.

THE USE OF SWINE STOMACH IN PERNICIOUS ANÆMIA.

In September, 1929, Sturgis and Isaacs, working together, and Sharp made independent reports of clinical improvement in patients with pernicious anæmia as a result of the ingestion of desiccated swine stomach. In view of the success already obtained with both raw and cooked liver, it was at once recognized that the new therapeutic agent should be carefully investigated and its value determined. H. Milton Conner, of the Division of Medicine, the Mayo Clinic, has made a recent report on the subject.[1] He explains that he was influenced to try the effect of administering the raw and cooked stomach of a meat-producing animal to patients with pernicious anæmia by two facts. The first was that Castle and Locke had demonstrated that meat fed to normal persons and recovered by vomiting and then fed to patients with pernicious anæmia, produced the same effect on the blood as is produced in patients of this type by the administration of liver. The second was that practically all patients with pernicious anæmia suffer from achlorhydria and that 26% of a large series of relatives of persons with pernicious anæmia have been found to suffer from achlorhydria. Conner's series of patients included only eleven persons. Ten of these were fed on raw and one on desiccated stomach. Unfortunately five of them had to be excluded from consideration in evaluating the results of treatment for various reasons. The average length of stay of these patients in hospital was 27·1 days. The average increase of erythrocytes per week while the patients were in hospital was 255,700. This increase was not so great as that found in a larger series in which treatment included liver diet or liver extract or a diet rich in vitamins with a small amount of liver. It is suggested that the discrepancy is due to the smallness of the series. To none of the patients was hydrochloric acid given and in five who were tested there was no return of free acid.

Conner is concerned chiefly as to whether raw swine stomach is more effective than liver as a therapeutic agent. He states that more work will have to be done before this can be determined. This is undoubtedly correct. Even if one of these animal tissues is slightly more effective than the other, the more plentiful tissue will be used. It must be noted in this connexion that it has not been determined whether the whole stomach or part of it is effective. Conner regards it as probable that the fundal mucous membrane is the effective portion because it secretes hydrochloric acid and gastric ferments. He does not go beyond this in his discussion. More extensive observations of this kind may possibly throw some light on the causation of pernicious anæmia. Liver and stomach both contain an effective substance. These two substances are not necessarily identical in form. If it could be shown that the hydrochloric acid of swine's stomach was essential to successful treatment with that organ, it might be found that the hydrochloric acid acted on a substance in the stomach which is the precursor of the effective substance found normally in liver. The observations of Castle and Locke, previously mentioned, do not make this improbable.

[1] *The Journal of the American Medical Association*, February 8, 1930.

Abstracts from Current Medical Literature.

MEDICINE.

Gastro-Duodenal Ulcer.

J. L. ROUMAILLAC (*Journal de Médecine de Bordeaux*, May 20, 1929) describes a new treatment for ulcer of the stomach. Jarny considered that any defect of duodenal regurgitation favoured the formation of gastric ulcer and surgeons thought that duodenal reflux was necessary for good results in surgical treatment of gastric ulcer. Following his own researches on gastric acidity and on the blood cholesterin content in various diseases, Jarny introduced his treatment for gastro-duodenal ulcer with a preparation the basis of which is a substance allied to cholesterin. Rosenberg also recently described a method of treating this condition by using cholesterin and bile salts. Jarny denies the existence of antipepsin and states that the bile salts and bile amino-acids prevent autodigestion of the stomach. One of these amino-acids, glycocol, combines in the liver with an acid, cholic acid, to form glycocholic acid, which the liver cells pour into the bile and hence into the intestine. By this means the duodenal fluid protects the gastric mucosa against acidity. In the course of digestion the stomach may suffer erosions due to trauma, the effect of hard or partly chewed food. The erosions heal quickly in animals, so long as duodenal regurgitation is satisfactory; but when an alteration in this regurgitation occurs owing to abnormal movements of the stomach or duodenum as a result of nervous influences, these erosions may persist and lead to ulcers. Volhard showed that the same occurs in man. Cholesterin is present in all cells and fluids of the body; the suprarenal glands are said to regulate the amount in the blood. Food affects the content of cholesterin in the blood and the bile. The red and white blood cells contain large quantities of cholesterin which is antihæmolytic and fixes hæmolytic toxins, bacteria and endogenous or exogenous toxins, including those from foci of infection and alimentary toxins. When the cholesterin content of the blood is insufficient or the toxins are in excess, the latter pass into the blood stream and by acting on the vagus and sympathetic alter the secretion and motility of the stomach so that hyperacidity occurs and the duodenal reflux is inhibited. The result is the formation of an ulcer. This is the theory advanced by Jarny, that hypocholesterinæmia leads to gastric ulcer and that deficient duodenal regurgitation has the same effect. He instituted a method of treatment with bile and cholesterin by mouth; these were covered by glyzine or glycyrrhizin as an excipient, as these substances dissolve in the stomach. A preparation called "Salvacid" fulfils the requirements. This contains dried ox bile, glycocholic acid and extract of a sap, ten centigrammes of each, with glycocoll or glycyrrhizin as much as is required as an excipient. This is given as compressed tablets four to twelve times a day, two after each meal as a rule, with a large drink of warm fluid. The sap contains a substance, thujone, which is allied to sabinol, a constituent of cholesterin. A diet of cream, in small quantities frequently, is recommended with this treatment. The author quotes 210 patients treated by Arany at Carlsbad. He himself treated a small number of patients with definite relief of symptoms.

Blood Phosphate in Nephritis.

J. B. NAKADA (*Journal of Experimental and Clinical Medicine*, August, 1929) discusses the significance of inorganic phosphate in the blood plasma in nephritis. The method of determining phosphate percentage is described. Eighty patients were investigated. It was found that in chronic nephritis and uræmia the blood phosphate percentage was usually high, at any rate towards the terminal stages (five to fifteen milligrammes per hundred cubic centimetres). In uræmia the high blood phosphate content usually corresponded with high blood urea, fifty milligrammes or more, but when the blood urea was above fifty milligrammes and the blood phosphate less than five milligrammes the prognosis was less grave. In other words, high blood phosphate *plus* a high blood urea content was more serious than a high blood urea content alone and in the patients studied usually indicated an early fatal termination. In other medical ailments the blood phosphate was usually between two and four milligrammes per hundred cubic centimetres.

Hyperparathyreoidism.

R. M. WILDER (*Endocrinology*, May-June, 1929) describes a case of hyperparathyreoidism or tumour of the parathyreoid glands, associated with *osteitis fibrosa*. It has been shown that Collip's parathyreoid extract in animals causes an increase in blood calcium and a decrease in blood phosphates with increased excretion of calcium and phosphorus, thus producing a negative balance of calcium and phosphorus. Since tetany is a sign of parathyreoid deficiency, hypotonicity might be a sign of overaction of the parathyreoids. Forty-seven cases of enlargement of the parathyreoids were reviewed by Hoffheinz. In twenty-seven of these diseases of the skeleton were noted, *osteitis fibrosa*, osteomalacia and rickets. A few cases have been reported in which removal of the parathyreoid glands or of a tumour of the parathyreoids has been followed by decreased blood calcium and improvement clinically in cases of *osteitis fibrosa*. The author's patient was a woman, aged thirty-two, who had always been weak and nervous, had neglected to take vegetables, milk or cream and had indulged in sweets. In 1921 pain in the right hip began, in 1923 a lump was noted in the thyreoid gland. In 1925 she had a swaying gait, was sallow and had lost all but four teeth. A spherical mass, three centimetres in diameter, was noted at the lower pole of the right lobe of the thyreoid. X rays revealed rarefaction of the pelvic bones without deformity. Muscular weakness developed. A year later cystic areas of rarefaction above the condyles of the right femur were noted, there was pain at this site and an operation was performed with removal of a giant cell tumour. The blood calcium was slightly increased at several examinations, 11·6 to 12·8 milligrammes per hundred cubic centimetres. A parathyreoid tumour was removed and blood calcium fell in three days to eight milligrammes; calcium excretion fell considerably. Calcium phosphate was given by mouth and the patient improved rapidly. Three months later X rays revealed increased density of bone due to calcium deposit and the blood calcium was eight milligrammes per hundred cubic centimetres.

The Significance of Glycosuria.

J. E. PAULLIN (*Annals of Internal Medicine*, August, 1929) in discussing the significance of glycosuria, states that when a reducing substance is found in the urine two questions immediately arise: (i) Is the reducing substance glucose? (ii) If the reducing substance is glucose, what condition is responsible for its presence? He discusses the question of whether glucose is present in the urine of normal persons and decides that at present the weight of evidence is that glucose *per se* does not seem to occur in normal urine. It is well known that other substances such as lactose, maltose, pentose and lævulose give positive results to reduction tests. These substances can be differentiated from glucose by various tests such as the fermentation test and the phenylhydrazine test. Such substances as uric acid and creatinin which occur in concentrated urine can be differentiated by diluting the urine and again testing. Chloroform, used as a preservative, may give a positive reduction. The conjugated glycuronates which are found after the administration of certain drugs (such as chloral, turpentine, camphor, menthol and phenol), do not ferment with yeast and do not form osazone crystals with phenylhydrazine. Glucose produces a typical reaction with Benedict's solution, readily ferments with yeast, is dextrorotary and forms characteristic osazone crystals with phenylhydrazine. When it is determined that glucose is present, it is necessary to discover the nature of the disturbance causing its presence. Firstly, several specimens of urine may be tested to determine whether glucose is constantly present, secondly, the blood sugar during fasting may be determined and again two

hours after an ordinary carbohydrate meal and thirdly, the glucose tolerance test may be used. The author utters a word of warning that a normal person who has been on a restricted carbohydrate diet for any length of time, will respond with hyperglycæmia and glycosuria. The results of the glucose tolerance test place the individual in one of several groups. The first of these groups is renal glycosuria, a condition in which the renal threshold is low. Patients with this condition store, utilize and metabolize glucose in a perfectly normal manner. The second group is cyclic renal glycosuria or alimentary glysuria. In this condition, an otherwise normal individual excretes urinary sugar after partaking of food rich in carbohydrate. This may be due to an abnormal post-prandial hyperglycæmia or a low renal threshold. It is not definitely settled whether these patients are perfectly healthy. The alimentary glycosuria may be due to insufficient insulin production. The third group is *diabetes innocens*. In this condition certain characteristics of both *diabetes mellitus* and renal glycosuria are found. Sugar is constanly excreted in the urine and there is an increase in glycosuria with an increased carbohydrate intake. There is no disturbance of the ability of the patient to metabolize, store and utilize carbohydrate. At the end of three hours, the blood sugar returns to normal. The fourth group is *diabetes mellitus*. In this condition, there is inability to metabolize, store and utilize carbohydrate. When it has been determined to which group the patient belongs, appropriate treatment can be instituted.

The Clinical Significance of Low Basal Metabolic Findings.

C. W. WARREN (*The Clifton Medical Bulletin*, July, 1929) discusses the clinical significance of low basal metabolic findings in a series of 5,000 consecutive basal metabolic estimations. The patients selected were those in whom the basal metabolic percentage was 20% below normal. The calculation was based on the method of Aub and Du Bois and the instrument used was a portable Benedict apparatus. One hundred and fifty-two patients were accepted. In twenty-one of these an operation had been performed. The remainder of the group suffering from medical conditions, comprised one hundred and twenty-one patients; one hundred and eight of these were females and twenty-three males. The majority of the patients were in the fifth and sixth decades. The lowest reading was – 41%. Contrary to expectations a large number of underweight individuals were included in the group, sixty-four being underweight and seventy-seven overweight. Fatigue was the chief complaint of the majority. Slight anæmia was frequently noted. Low temperatures and low pulse rates were infrequent. Hypochlorhydria was found in many of the patients. Myxœdema was diagnosed in forty-eight patients, hypothyreoidism in forty-three and nervous disorders such as psychasthenia, psychoneuroses and hysteria were noted in thirty-three instances.

The Coronary Problem.

A. R. ELLIOTT (*Annals of Internal Medicine*, September, 1929) states that pathological change in the coronary vessels of sufficient extent and degree to bring about cardiac death may exist without attracting attention by unequivocal signs and symptoms. The coronary vessels may be found to be diseased when all other accessible parts of the vascular system appear unaffected. Disease of the coronary arteries is one of the most important causes of myocardial weakness. The later in life obliterative changes in the coronary vessels are postponed, the better they are tolerated. The prognosis, therefore, stands in inverse ratio to the age of the patient. From the diagnostic point of view the clinical evidence of coronary disease is found in anginal attacks, while the electrocardiogram may show indications of more or less severe muscle damage. During recent years chronic myocarditis, the failing heart of hypertension and certain of the adult arrhythmias have been regarded as being due to coronary disease. *Angina pectoris* is a manifestation due to ischæmia of the myocardium and generally arises when the circulatory system is embarrassed by undue strain. Anginal attacks, however, do arise during sleep. In certain cases paroxysmal tachycardia may occur. The differentiation of the manifestations of coronary disease from gall bladder, gastric or duodenal disease may be difficult. The record of a patient apparently suffering from coronary disease is quoted by the author to illustrate the diagnostic and therapeutic difficulties of the problem. He concludes by stating that careful clinical study with special care in obtaining all the details of the history aided by routine electrocardiography is the best method of attacking the problem.

Diabetes and Tuberculosis.

LÉON BERNARD (*La Presse Médicale*, December 9, 1929) points out that modern methods of diagnosis and treatment have greatly lessened the classical prognostic gravity of the combination of diabetes and pulmonary tuberculosis. Formerly, no less than 40% of diabetics died from tuberculosis; recent figures show this complication to be the cause of death in less than 10% of diabetics. Though he is not prepared to assign any particular reason, the author admits the frequency of concurrence of these diseases. He also stresses the fact that the tuberculosis tends to the exudative type, with a caseating pneumonic spread, notwithstanding that symptoms are often in abeyance. Radiography has revealed widespread unsuspected lesions in persons whose general appearance remained good. In the apparent conflict which arises in the treatment of patients afflicted with these two maladies, the author pleads for a rational plan of campaign. Superalimentation he condemns as achieving nothing but an aggravation of the diabetes. Careful dietetic restrictions are absolutely necessary and there is no truth in the objection that "Insulin" therapy has an unfavourable action on the course of the tuberculosis. For the latter there is need for earlier diagnosis. By this means the lesion may be found to be still unilateral and in these circumstances there is available a valuable weapon in artificial pneumothorax; this should be instituted without a moment's delay. If the lesion has become bilateral, the outlook is more serious. Salts of gold which the author has found to be of help in simple pulmonary tuberculosis, have had no effect in tuberculosis complicating diabetes. He concludes by emphasizing the great improvement in prognosis in these cases which has resulted from the employment of the therapeutic triad, regulated diet, "Insulin" therapy and artificial pneumothorax.

Alastrim.

E. LESCHKE (*Münchener Medizinische Wochenschrift*, December 13, 1929) discusses the recent outbreaks of smallpox and alastrim in Europe and America. He describes in detail the diagnostic points in both diseases and refers to the value of vaccination. In his opinion alastrim is a mild form of true variola with slight variations in the clinical picture. The mortality is not above 1% of the patients affected. The virus presents all the characteristic reactions of true smallpox and vaccination protects against both diseases. The same epidemiological measures must be adopted to stamp out both diseases. In order to lessen the risk of encephalitis following vaccination he recommends the use of a less virulent vaccine, vaccination as early as possible and the use of at most two skin incisions.

Congenital Syphilis.

F. STRUNZ (*Deutsche Medizinische Wochenschrift*, December 20, 1929) gives his results with "Myosalvarsan" in the treatment of congenital syphilis. Intragluteal injections of 0·3 gramme dissolved in two cubic centimetres of distilled water were employed. In children of two or three years, 0·2 gramme per kilogram body weight was given up to a maximum of 3·0 grammes. The length of a course was twelve weeks. In general injections were given every week, although with the use of small initial doses these could be given twice a week at first. There is very little local reaction and the only general reaction is a transient rise of temperature. Great care must be exercised in young babies with severe visceral syphilis. After two or three injections cutaneous lesions disappear and the children rapidly gain weight, have a good appetite and the

stools remain normal. In about 50% of the patients the serum reaction fails to occur—a similar proportion to that seen among those treated with "Salvarsan." After an experience of fifty cases the author strongly recommends "Myosalvarsan" especially for ambulatory patients.

Yellow Fever.

CORNELIUS B. PHILIP (*American Journal of Tropical Hygiene*, July, 1929) makes a preliminary report of the experimental transmission of yellow fever by several species of Aedes. Bauer (1928) has shown that *Aedes leukocephalus* (Newst), *Aedes (Aedimorphus) apicoannulatus* (Edw.) and *Eretmopodites chrysogaster* Graham, all African species, are capable of transmitting experimental yellow fever to *Macacas rhesus*. Specimens of *Aedes vittatus* (Bigot) were fed on animals suffering from yellow fever and at periods varying from eleven to thirty-nine days afterwards were allowed to bite six normal rhesus monkeys. Five of these died of yellow fever, while the sixth recovered after a sharp febrile period. Of three monkeys bitten by *Aedes africanus* (Theo.) from twelve to twenty-one days after the latter had fed on blood of sufferers from yellow fever, two died of yellow fever while the third suffered no ill effects, but died of yellow fever after having been inoculated subcutaneously with an emulsion of five macerated insects from the same batch. Three monkeys died of yellow fever after having been bitten by specimens of *Aedes simpsoni* (Theo.) which had been fed on infected animals nineteen, twenty-four and thirty days previously. One monkey bitten by these mosquitoes developed no symptoms of yellow fever and afterwards suffered no ill effects from the injection of known infective material. It was assumed to be immune. The importance of these three species of Aedes in the epidemiology of yellow fever has yet to be determined.

Hepatic Disease.

I. S. RAVDIN (*The Journal of the American Medical Association* October 19, 1929) discusses some aspects of carbohydrate metabolism in hepatic disease. In diseases of the biliary apparatus dextrose is often given intravenously before and after operation. The reason for this is that when the liver is diseased, its main function of storing carbohydrates in the form of glycogen and supplying the blood with sugar from this store may be interfered with. The ingestion of glucose by patients with liver diseases causes a higher and more prolonged glycæmia than in normal subjects. This indicates that the formation of glycogen in the liver may be diminished in such patients. It has been shown that when the common bile duct is ligated in dogs within two to six weeks, there is a diminution of glycogen stored in the liver. Davis also proved that chloroform can destroy one half of the liver tissue in a fasting animal and that with a high carbohydrate diet this liver tissue can be repaired in seven to nine days. Similar regeneration of the liver has been shown in dogs; in them liver destruction has followed ligation of the common bile duct and cholecystectomy. The author considers that isotonic or slightly hypertonic glucose solutions are more valuable in this respect than solutions of higher percentage and that a slow administration is the most useful, for in rapid administration or with high percentage of glucose much is lost through the kidneys. In liver disease as a rule the blood sugar is normal, hence there is no evidence that the pancreas is diseased and consequently no theoretical reason for giving "Insulin" with glucose; further it has not been proved experimentally that "Insulin" assists the storage of glycogen in the liver in these circumstances. When diabetes coexists with liver disease the use of "Insulin" together with glucose is indicated. Both chloroform and ether cause hyperglycæmia, followed by lessened glycogen in the liver. It is probable that dextrose is more valuable in preventing hæmorrhage in jaundice than is calcium; there is no evidence that blood calcium is diminished in jaundice. Intravenous injection of dextrose and a high carbohydrate diet are indicated before and after anæsthesia in liver disorders.

Cisternal Puncture.

A. E. BENNETT (*The Journal of the American Medical Association*, October 5, 1929), discusses the diagnostic and therapeutic indications for cisternal puncture. The patient is placed on one side as for lumbar puncture, with a pillow under the head to secure alignment of the cervical vertebræ with the lower part of the spine, the chin is well flexed. A small needle (number 20) is used, the left forefinger is placed on the spine of the axis, the needle lined up with the external auditory meatus and the glabella and pushed quickly through the skin; it is then pushed cautiously forward and upwards. If the puncture is in the midline, bony resistance will not be met or the needle may be made to strike the occiput and follow its border. After the depth of three centimetres has been reached the operator should proceed cautiously until the same feeling of giving way is felt as in lumbar puncture. If he is in doubt he should keep pulling the stylet out as the needle is inserted deeper. The *cisterna magna* is reached at a depth of 2·0 to 3·5 centimetres in infants and four to five centimetres in adults; occasionally it is six centimetres deep. The needle may be scratched at six centimetres to prevent its going deeper. The *cisterna magna* is 2·5 to 3·0 centimetres in depth at its upper level and 1·0 to 1·5 centimetres at the lower border. Ten patients with meningitis were treated, mainly by cistern puncture and administration of serum by this route. The pneumococcus was the suspected cause in one instance; all others were apparently meningococcal. Two patients died; one had tuberculous as well as meningococcal meningitis. When the spinal fluid was fibrinous, Ringer's solution was used to irrigate the subarachnoid space; the solution was washed in and out through the cistern needle and also between the lumbar and cisternal needles. When the spinal fluid is under great pressure, drainage should be carried out very slowly. Concentrated serum is recommended, as ten cubic centimetres are equivalent to thirty cubic centimetres of ordinary serum. Cistern puncture is advocated for routine treatment in meningitis.

Blackwater Fever.

W. M. HEWETSON (*Journal of Tropical Medicine and Hygiene*, June 15, 1929) discusses the symptoms of blackwater fever and suggests a method of treatment by operation. He challenges the statement that the liberation of hæmoglobin is the cause of the toxic symptoms in blackwater fever, contending that these are due to bile. In support of his argument he states that there is a latent period of some two to twenty-four hours from the first appearance of hæmoglobinuria until the development of icterus, during which the patient feels comparatively well. Serious symptoms are not evident until bile pigments are present in the circulating blood. In support of his argument that the icterus in blackwater fever is due to bile, he draws analogies with yellow fever. He quotes Thomson as stating that the gall bladder of persons who had died of blackwater fever, was in all cases filled with a dark viscid-looking bile causing obstruction to the biliary passages Of twenty-one blackwater fever patients investigated by Ross, the three who suffered from suppression of urine, gave a direct positive reaction to the Van den Bergh test, while in the remaining eighteen the result of the test was indirectly positive. Hewetson recommends that when there is suppression of urine during blackwater fever, cholecystostomy should be done under local anæsthesia. When the chance of life is remote, a large intravenous injection of saline solution should be given, followed by cholecystostomy in four hours if no definite improvement is manifested.

Thromboangiitis Obliterans (Buerger's Disease).

A. M. RECHTMAN (*Medical Journal and Record*, April 3, 1929) reviews the important features of *thromboangiitis obliterans*. Definite signs are not found in the early stages of this disease. The symptoms are generally manifested first in the lower extremities; they begin in one and soon involve the other. The upper extremities may be involved later. The usual age incidence is between twenty and fifty years. The majority of patients seen by the author were less than thirty-nine years of age; 99% were males and over 50% were Hebrews. The disease is not uncom-

mon in China, Japan, Korea and Turkey. The sufferers from the disease are usually large users of tobacco. Exposure to cold and trauma of the extremities in one otherwise predisposed are added factors. The exact ætiology is unknown. With regard to the pathology, the lesion was described by Buerger as an inflammatory process affecting the arteries and veins with the presence of giant cells and thrombosis in the acute stage. At a later period, the thrombus becomes organized and canalized and the products of inflammation are replaced by a periarterial fibrosis, the arteries and veins being bound in a dense adherent mass. The first symptom complained of is cramp in the leg caused by exercise and relieved by rest. This is due to deficiency of the blood supply. Coldness of the hands is usually a constant complaint. Colour changes are important, the feet becoming red when dependent and pale or blanched when elevated. Ulcers due to trauma may appear and lead to gangrene. The absence of the pulsations of the *dorsalis pedis* artery should give rise to suspicions of the presence of *thromboangiitis obliterans*. Sometimes the posterior tibial, less frequently the popliteal or even the femoral pulses, cannot be palpated. The degree of impairment of the circulation may be further tested by means of the oscillometer and the intradermal injection of normal saline solution. The clinical course of *thromboangiitis obliterans* may vary considerably, remissions being not uncommon. With regard to the differential diagnosis, it is important to remember that circulatory disturbances fall into two groups, the functional vasomotor disorders and the organic circulatory diseases. Of the functional disturbances, Raynaud's disease (vasoconstrictor type) and erythromelalgia (vasodilator type) are the most important conditions, while arteriosclerosis is the chief organic circulatory disease from which *thromboangiitis obliterans* must be differentiated. The distinction between arteriosclerosis and *thromboangiitis obliterans* is sometimes difficult, but the age incidence (arteriosclerosis occurs at fifty years of age and upwards, *thromboangiitis obliterans* from twenty to fifty) and the distribution of the disease (general in arteriosclerosis, local in *thromboangiitis obliterans*) may help in deciding this point. X rays may be of value in demonstrating calcareous patches in the arteriosclerotic vessels. The functional types are usually present in females. A functional arteriospasm sometimes simulates Buerger's disease.

Tularæmia.

P. R. WITHINGTON (*New England Journal of Medicine*, September 26, 1929) in reporting the history of a patient suffering from tularæmia, discusses the salient points of the disease. Tularæmia is caused by *Bacillus tularense*, a small Gram-negative, non-motile, non-spore-bearing organism. It is aerobic, ferments glucose, lævulose, mannose and glycerol, forming acid, but no gas. The disease occurs in wild rodents. It is transmitted to man directly from the animals or through the bites of deer-flies or ticks. The infection reported was apparently the first recorded instance of the disease in New England, United States of America, but instances of the disease are frequent in other parts of America and also in Japan. Four clinical types of the disease are described. The first is the ulcero-glandular type. The primary lesion is a papule which later becomes an ulcer and is accompanied by regional glandular enlargement. The author's patient suffered from this type of the disease. The second is the oculo-glandular type in which the primary lesion is conjunctivitis. The third type is characterized by enlargement of regional lymphatic glands without ulceration, while the fourth type is the typhoidal; in this there is no glandular enlargement and the diagnosis is made only by serological examination. While the incubation period may vary from twenty-four hours to nine days, the average is three days. The onset is sudden and is characterized by headache, vomiting, chills, aching bodily pains, sweating, prostration and fever. Usually there is an early remission of temperature and freedom from symptoms, but the symptoms return with the rise in temperature. In about half the cases the glands break down, but in the other half, they remain hard and tender for about three months and gradually return to normal. Suppuration has been noted ten months after the onset of the disease. Convalescence is slow, two to three months being the usual duration. Relapses lasting six to eight days have occurred eight and ten months after the onset. In the case reported the organisms were not recovered from glands, but the diagnosis was justified by the history of repeated exposure to the bites of ticks, the clinical course of the disease and the serological reactions.

Blackwater Fever in Malaya.

G. WAUGH SCOTT (*Malayan Medical Journal*, September, 1929) discusses blackwater fever as it occurs in Malaya. The absence of blackwater fever in a malarious country such as Malaya has been used as an argument against the malaria theory of the causation of the disease. But blackwater fever does occur in Malaya and appears to merit more attention than has hitherto been accorded it. In the author's experience every blackwater fever patient has previously suffered from improperly treated malaria. Whatever might be the actual cause of the hæmoglobinuria, there can be no doubt that malaria is present and should be treated. Scott is convinced that his results have been better since discarding Bastianelle's rule of giving quinine only if malaria parasites are found on examination of the blood. He injects 0·7 gramme of quinine bihydrochloride into the buttock night and morning. As there is always some degree of collapse and lowering of blood pressure, 0·25 cubic centimetre each of adrenalin and pituitrin is given twice a day. There is some similarity between the collapse in blackwater fever and the collapse in cholera and this led to the intravenous administration of hypertonic saline solution (568 cubic centimetres twice a day) with satisfactory results. These injections appear to be the only effective agent in preventing total suppression of urine—the complication most to be dreaded in this disease. Sometimes the administration of the saline solution is followed by a rigor which should not cause anxiety. Sternberg's mixture is useless and even harmful, as these patients with badly damaged kidneys are unable to eliminate all the mercury.

Erysipelas.

H. B. CUSHING (*Canadian Medical Association Journal*, September, 1929) discusses erysipelas in childhood. Eighty patients were studied, thirty-nine were under one year old. Three groups were noted, facial, limb and trunk erysipelas; the latter was most serious. Several strains of streptococci caused erysipelas, the contagiousness of which was rather like that of pneumonia. Erysipelas often was superimposed on other conditions involving as a rule an open wound. Treatment varied enormously, but the results were much the same with all forms of treatment. The author concludes that no drug influences its course and that local treatment will not prevent its spread; polyvalent antistreptococcal serum had few successes, blood transfusion and the use of the serum of convalescents were disappointing, as was the latest specific antistreptococcal serum introduced by Birkbang and Amos. In the author's opinion it is best to treat erysipelas as a self-limited infection and to nurse the patient as for pneumonia, applying only a bland lotion to the face and not expecting too much of drugs and sera.

Protection against Bacillary Dysentery.

W. WALKER and R. C. WATS (*Indian Journal of Medical Research*, July, 1929) report the failure of oral bilivaccine as a protective agent against bacillary dysentery in their experiments at Poona and Secunderabad. Using a bilivaccine prepared by the Biotherapie Compagnie, Paris, according to the researches of Besredka, the authors attempted to immunize one thousand four hundred soldiers immediately prior to the commencement of the dysentery season. One tablet of vaccine was given on three successive mornings on an empty stomach, food being withheld for one hour after each treatment; twenty-eight or 20% of the vaccinated suffered attacks of definite bacillary dysentery during the dysentery season, while of the 3,680 unvaccinated, 52 or 14·10% contracted the disease. The clinical course of the disease did not appear to be modified in individuals who had been vaccinated.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Darling Building, University of Adelaide, on November 28, 1929, DR. H. GILBERT, the President, in the chair.

Urology.

DR. IVAN B. JOSE read a paper entitled: "Notes on Urological Surgery Gleaned Abroad" (see page 383).

DR. W. JOHN CLOSE read a paper entitled: "Preoperative and Postoperative Management in Urology" (see page 386).

DR. B. SMEATON thanked the speakers for their papers. He agreed with Dr. Jose that the routine examination, including the preparation of pyelograms where necessary, should be completed at one cystoscopic examination to save unnecessary inconvenience. He also emphasized the importance of full investigation in hæmaturia.

DR. MALCOLM SCOTT asked what was the effect on the kidney of repeated persistent attacks of pyelitis.

DR. H. GILBERT asked if tuberculin was of much benefit in tuberculosis of the genital urinary organs, also in what proportion of cases was hæmaturia due to chronic nephritis.

DR. OWEN MOULDEN said that he had had two patients with pyelitis who had apparently been cured by oral administration of mercurochrome and asked if this method was widely used.

DR. C. DUGUID, speaking of X ray investigation of the kidney after it had been delivered, mentioned a case occurring at the Mayo Clinic in which a second stone was found by the surgeon only after much persistence on the part of the radiologist. But for this examination that stone would have been left in the kidney.

He regarded the preoperative treatment of patients before prostatectomy as an essential routine and he used the sacral caudal method of anæsthesia with subcutaneous blocking of the lower part of the abdomen. Hunt's operation, open from the start, was preferred to Sir John Thomson-Walker's which at the beginning was blind.

The necessity for a pyelogram in the urological examination was indicated in a patient with pyelitis whom he had seen two years previously. There had been double ureter, the urine from one giving a pure culture of *Bacillus coli* and that from the other a pure culture of staphylococci.

Dr. Close in reply said he had not seen mercurochrome given by mouth. Mercurochrome was recognized as being more effective for coccal than for *Bacillus coli communis* infection. He also said that some urologists, Thomson-Walker in particular, were very keen on tuberculin as a postoperative treatment. Tuberculin, T.R., was favoured and the initial dose was as small as one ten-thousandth of a milligramme, increasing gradually to one-fifth of a milligramme in twelve months. The effects of persistent or repeated attacks of *Bacillus coli communis* pyelitis were mainly seen in the calyces, namely, a blurring of the outline up to a clubbing or definite dilatation. If a pyelogram were taken and revealed dilatation, he thought some other treatment than medicine would be indicated.

Dr. Jose replied that he had not had any experience of mercurochrome given by the mouth, but that he had seen it used at the Mayo Clinic in acute urinary infections, as occurred after instrumentation, by intravenous injection in dilute solution with good effect. He was in the habit of using tuberculin as an adjunct to the surgical treatment of renal tuberculosis. Its use was generally favoured by most urologists. He could not say what proportion of hæmaturia was due to granular kidney.

Cerebral Abscess Complicating Otitis Media.

DR. H. M. JAY showed a patient, W.H., a female, *ætatis* fifteen years, who had been admitted to the Adelaide Hospital on April 10, 1929, complaining of pain in the right ear for five days. The temperature had been 38·3° C. (101° F.), the pulse rate 104 and the right ear discharging profusely. The ear had been treated by inserting gauze wicks and an ice bag had been applied to the mastoid. The temperature had come down for two days, then an evening pyrexia had developed. Tenderness and pain had almost disappeared four days after admission.

Cultures had contained *Staphylococcus aureus* and an X ray examination of the mastoid had revealed a haziness of cells around the base of the right petrous bone.

On April 24, 1929, the patient's condition had been greatly improved, the temperature normal and the discharge less. Four days later she had become slightly drowsy and had vomited once or twice after meals. She had felt very well and had had no headache. The pulse rate had been 88.

On May 2, 1929, she had still felt well, though drowsy, had had no headache and the pulse rate had been around 84, the lowest being 70. Optic discs had had blurred margins, cerebro-spinal fluid had been clear and under increased pressure. There had been no neurological signs and the fields of vision had had no quadrantic defect.

Dr. Jay had decided that the mastoid and temporo-sphenoidal lobe should be explored and the next day operation had been performed. The disease in the mastoid had been limited to the region of the antrum; the *dura* of the middle fossa had been inflamed and on exploration of the brain about forty-five cubic centimetres (one and a half ounces) of pus had been evacuated. Attempts at culture from the pus had yielded no growth of organisms. The patient had made an uninterrupted recovery.

Dr. Jay called attention to the mild degree of the symptoms present, namely, slight drowsiness and two or three attacks of vomiting, considering the size of the abscess and remarked that there was no headache or fall in the pulse rate. He drew the attention of those present to the use of the long Killian nasal speculum as a means of aiding the evacuation of pus from a cerebral abscess without inflicting undue damage on the brain.

Malunited Fracture.

DR. L. O. BETTS showed two patients illustrating the use of the Thomas wrench in malunited fractures. The first had suffered from a Pott's fracture with complete backward dislocation of the foot carrying with it the posterior lip of the tibia. The dislocation had been reduced three weeks after its occurrence after the *tendo Achillis* had been lengthened by plastic tenotomy with a tenotome, followed by refracturing the fibula and increasing the deformity with a Thomas wrench. The patient at the time of the meeting, twelve months afterwards, had excellent function of the joint with almost a full range of movement.

The second patient had suffered from a double Colles's fracture, also seen three weeks after the injury with malunion in both. One fracture had been compound and become infected, had a sinus involving the flexor tendons in front of the wrist; they were contracted. After the sepsis had subsided, the wrist and fingers had gradually been stretched out into extension by splinting, daily exercises being given. A good functional result had been obtained, but considerable deformity remained. The deformity in the other wrist had been reduced with a small Thomas wrench. A perfect functional and anatomical result was obtained. In both arms there was median nerve irritation which was very persistent and delayed treatment in the infected arm.

Huntington's Chorea.

DR. A. R. SOUTHWOOD showed a patient, a man of forty-eight years, presenting the main features of Huntington's chronic progressive hereditary chorea: the choreiform movements, the onset in middle life, progressive mental weakness and the familial and hereditary incidence of nervous disorder.

The man had been in apparently normal health till two years previously. Mental changes had then begun to appear; he had become restless, had not been able to give

a coherent history of himself and had shown a lack of intelligent interest in his environment. The choreiform movements had appeared first about eighteen months before. The hands and face had first been affected and the rest of the body later. The facial movements were almost constant and were aggravated by excitement. The speech was rather indistinct and blurred. The gait was of an incoordinate lurching character. The deep reflexes were exaggerated and the contraction response persisted longer than normal. The patient's mother had been of nervous temperament and had died in middle age. One brother was suffering from dementia and had been in a mental hospital for some months; he had not manifested any choreiform movements.

LISTS OF MEMBERS.

THE lists of members of the several Branches of the British Medical Association in Australia are now available. Copies can be purchased from the office of THE MEDICAL JOURNAL OF AUSTRALIA at one shilling each.

Correspondence.

THE REDUCTION OF MATERNAL MORTALITY.

SIR: In Dr. E. R. Roseby's letter in the last issue of your journal is set out in plain terms his grave concern at the humiliating position of himself and his fellow practitioners, clearly revealed by the latest reports concerning maternal morbidity and mortality. He earnestly pleads for inquiry into the reasons for the disparity between the records of doctors and midwives and his appeal is highly commendable. It is a pity that his first suggestion is not founded on fact. The vagina rarely harbours sepsis. Dr. Roseby's statement to the contrary is easily proved to be illogical, otherwise we could not expect this factor to be negligible in the practice of midwives. To him autoinfection is of prime importance, but not to nurses, which is absurd. He does them as a body great injustice by charging nurses with ignorance of "the science of presentation." He is entirely in error in supposing that a skilled maternity nurse never at any stage palpates the *os uteri* or the lower cervical canal. Abdominal palpation will not eliminate the tragedy of the prolapsed cord, for instance.

The real reason for the better results in the practice of nurses lies in the fact that they rarely come in contact with obvious sepsis from which is derived virulence. Furthermore, nurses are accustomed to use stronger detergents for their hands than do doctors, in spite of the more obvious need that they should. Doctors wear gloves, but the position remains unchanged since their introduction.

Like so many others, Dr. Roseby confuses ubiquitous germs of putrefaction (saprophytes and symbiotes) with pathogenic transplants, placing them all on the same plane of importance as causes of acute infections. Dirty surroundings and attendants to fit are not embarrassed by puerperal infections in the absence of contamination by any source of active virulence. Are there still amongst the members of our profession individuals who believe that diphtheria and sore throat arise from foul drains or that any infection arises from such a source? If all life comes from the egg, so as surely all microbic disease comes from eggs which we may designate as bad ones whose upbringing has fitted them with invasive powers, fleeting, but strong.

The anxious thoughts of the general practitioner had to find expression sooner or later and Dr. Roseby is to be congratulated as one of the first. It is quite seven years since I first ventured to suggest that our profession was heading for the rocks. We have just about struck them and the sorry part of the whole situation is that those on shore will only laugh at our plight. For instance, what will ninety and odd *per centum* of the women who are expectant mothers, think of the idea of the "new maternity service" that they should bear the pangs of labour without an anæsthetic? Dare we tell them that we must leave all this work to nurses because we do not know how to avoid carrying infection to them? Not only will there be laughter; there will be execration, disgust, contempt and a widespread loss of confidence.

If my sense of humour were not drowned in a deep compassion, I would join in the laughter. Finally, I will say that Dr. Roseby, like others who have quoted the Aberdeen report, did not read it carefully. Its authors were careful to eliminate all unfair comparisons. It is time we left off vilifying the nurse.

Yours, etc.,

A. C. F. HALFORD.

Brisbane.
February 15, 1930.

ANÆSTHESIA.

SIR: In the excellent letter of Dr. A. B. K. Watkins (January 13, 1930) appears the following statement: "In fact it is a farce to pretend that to give an anæsthetic with the modern one tap ether control is even as difficult as an open anæsthetic."

If we omit the words "farce" and "pretend" and give the ordinary meaning to "difficult," the statement needs revision. We are not concerned with the easiest or most difficult method, but with the safest. About this there are honest differences of opinion, but on strict examination it is found that the safest method is invariably the one we ourselves use and in which we have become adepts.

In my experience the only method of general anæsthesia, except for a few special cases, which is both easy and safe, is open ether for surgery and open chloroform for midwifery. Open chloride of ethyl fulfils all the necessary requirements for minor cases.

Yours, etc.,

J. F. MERRILLEES,
M.B. (Melb.), F.R.C.S. (Edin.).

St. Marys, Roma, Queensland.
February 16, 1930.

TONSILLECTOMY.

SIR: I have been interested and somewhat amused over the correspondence about enucleation of tonsils.

As in all surgical operations the technique should be suited to the case. To me it is ridiculous to use intratracheal anæsthesia and dissection for children. It is making a circus of what in experienced hands is a simple and rapid operation.

I have operated on somewhere between 20,000 and 30,000 cases without a death and without a secondary pneumonia. It is quite easy to shell out the tonsils under ethyl chloride or even gas anæsthesia, if the patient is sitting up, and remove the adenoids at the same time. I do many adults with the guillotine because it leaves a much better throat. There is no scarring. That is important, as one may have a dry throat if there is scarring and that is vital, especially for singers and speakers. For nervous adults I use intratracheal anæsthesia and dissection. For the average adult I use and prefer local anæsthesia preceded by a synergic anæsthetic which calms the patient and prevents gagging. I do most of these sitting up. It is easier, as you have the cooperation of the patient, who can expectorate. Lying down one has to use the sucker and it is much more uncomfortable for the patient. Sitting up, a tongue depressor is unnecessary after the injection of the local anæsthetic.

For full-blooded middle-aged people I prefer a local anæsthetic, as you can tie your vessels as you come to them. One can remove most buried tonsils with the guillotine, but it is wiser to give ether in case there is any difficulty. You have plenty of time to dissect any remaining fragment.

After all, the method you are most familiar with is generally the most efficient. I make it my practice to tie all bleeding points as in any other surgical operation. I believe some practitioners are expert at sewing up the socket. I have never tried it. It seems less surgical than tying the bleeding points.

May I offer one suggestion? When one has difficulty in securing the bleeding vessel, a small, rounded, moist gauze swab dipped in a mixture of powdered iron alum and formidin and packed into the socket will seal any bleeding point. This glues itself into position and can be left until the next day. I have never seen it fail. This is particularly useful in secondary hæmorrhage. It is well to hold a swab of cocaine and adrenalin in place for a few minutes beforehand. This dries the surface and the alum does not sting.

Finally, in these times of financial depression I think one should cultivate the least expensive method for the patient, provided it is efficient in your hands.

Looking back over many years of practice I shudder to think how one would have got through if one had been tied to a pantechnicon all the time.

Yours, etc.,

W. N. ROBERTSON.

"Craigston,"
217, Wickham Terrace,
Brisbane.
February 24, 1930.

AN EXPLANATION.

SIR: I notice in the issue of THE MEDICAL JOURNAL OF AUSTRALIA for February 15 a letter signed by three Brisbane doctors disclaiming any association with the publication of an article in *The Daily Mail* of January 25, 1930, under the title "Clinic for Brisbane."

This statement as it stands is a little unfair, as it may appear to readers of THE MEDICAL JOURNAL that the article mentioned was either inaccurate or misleading. Will you please, therefore, allow me to say first that the article did not mention any doctor's name or suggest that any doctor was "associated" with it and secondly that the accuracy of the article has not been challenged, nor do I think it can be challenged. The purpose of the letter apparently is to show that it was not written or inspired by any of the doctors signing the letter. That statement is perfectly accurate, even if superfluous, and I have pleasure in endorsing it.

Yours, etc.,

CHAS. G. SLIGO,
Editor.

"The Daily Mail,"
288-294 Queen Street,
Brisbane.
March 6, 1930.

SUDDEN DEATH AT GOLF IN A HYPERPIETIC.

SIR: Glenworth Reeves Butler wrote on April 24, 1925: "What is that mysterious factor which creates anger and rage in the breast of a golfer and causes his blood pressure to rise from one hundred and forty to nearly two hundred?"

Just a few weeks ago a patient of mine dropped dead whilst actually watching a drive he had made on the Roseville Golf Links.

I had seen him first in July, 1929, being called in to see him as he had influenza and was not very ill. On examination the temperature was 100° F., the pulse 85, but the blood pressure was 180 millimetres. I pointed out to him that he was a victim of thickened arteries and should avoid all sudden exertions and should cut down his meat eating.

At periods of four to six weeks he came and saw me and his blood pressure was very constant at 140 millimetres. He had been taking small doses of iodide of potash for ten days, omitting a week only in the month.

He went to see the doctor of the railway department, unknown to me, who rather gave him a good report and told him not to worry. He was a civil servant, a non-drinker and a non-smoker.

He called to see me on January 3, 1930, and his blood pressure was 140 and I sent him to bed for a few days.

On February 1, 1930, I was summoned to see him as he fell dead on the links after completing his drive.

In future I would advise any of my patients of nervous disposition and even a potential hyperpietic to turn to the ancient pastime of bowls instead of the modern game of golf.

Yours, etc.,

A. M. WATKINS, M.B., B.S.

741, Bancroft Avenue, Roseville,
New South Wales.
March 7, 1930.

ACUTE YELLOW ATROPHY OF THE LIVER.

SIR: I was very interested in the report of a case of acute yellow atrophy of the liver by Dr. F. A. Hope Michôd on page 296 of THE MEDICAL JOURNAL OF AUSTRALIA of March 1. Dr. Michôd is to be congratulated on his comprehensive survey of the literature and the comparison with delayed chloroform poisoning.

In *The British Medical Journal* of February 25, 1911, I reported two cases of death from post-anæsthetic acid intoxication. These cases had much in common with the ones mentioned by Dr. Michôd. The onset of symptoms was eight hours and twenty-five hours respectively. Vomiting occurred in both, but the most marked symptoms were rapid breathing, smell of acetone in the breath and marked acetone and diacetic acid in the urine. Later they became cyanosed, the pupils were dilated and coma came on. In one a partial autopsy showed no abnormality except a pale yellow liver.

In THE MEDICAL JOURNAL OF AUSTRALIA, September 14, 1918, I reported a case of severe post-anæsthetic acidosis with recovery. In this patient vomiting was marked, respiration was increased and the urine showed large amounts of acetone bodies. The condition was successfully combated by intravenous infusion of glucose and sodium bicarbonate. Two days later the liver dulness was found to be reduced by thirty-seven millimetres, but two months later had returned to normal.

Jaundice was not present in any of these patients nor was there any bile, albumin or sugar in the urine.

The lesson to be learnt seems to be: (i) avoid chloroform, (ii) test the urine for acetone bodies, (iii) give glucose.

Yours, etc.,

GILBERT BROWN.

36, Walkerville Terrace,
Gilberton, Adelaide.
March 10, 1930.

Obituary.

JOHN CORBIN.

BY the death of John Corbin in the prime of his life the medical profession in South Australia has lost one of its most striking personalities and disinterested leaders. He was born on June 1, 1878. He was the third son of

the late Dr. T. W. Corbin who for many years conducted a very large general practice in Adelaide and who moved the resolution in 1879 which led to the foundation of the South Australian Branch of the British Medical Association. He was educated at Saint Peter's College and Queen's School. At both schools he gave evidence of a brain much above the average. Indifferent health during boyhood and early adolescence prevented him from taking a leading part in athletics. After leaving school he worked for a time on the goldfields of West Australia and his health became robust. Through the generosity of an uncle he was enabled to proceed to England and entered Saint Bartholomew's Hospital Medical School. He showed a zest for Rugby football and played for the hospital fifteen. This was no mean achievement, for he had never played Rugby in Australia. At the end of the required course of study he took the M.R.C.S. (England) and L.R.C.P. (London). It was his wish to take the F.R.C.S. (England), but financial circumstances compelled him to return to Adelaide in 1903 and join his father in practice. When his father retired, he practised for a time in partnership with his brother, Dr. Cecil Corbin. He relinquished a large practice to go to the war in 1914 as major in the First Australian Stationary Hospital. Prior to the outbreak of the war he had served in the Australian Army Medical Corps. He saw service in Egypt and in the hope of getting nearer to the front he transferred at Lemnos to the First Australian Casualty Clearing Station and was present at "the landing." He did very valuable work on the "beach" until he was invalided back to Australia with enteric fever. Soon after his recovery he again volunteered for active service and was given command of the Third Australian Casualty Clearing Station, a unit of which he was very proud. He commanded the Clearing Station for a year in France, when circumstances compelled his return to Australia. Corbin's war service was most distinguished and though he was not decorated, those who served with him know that he amply deserved the honour.

At the conclusion of the war he was appointed Honorary Assistant Surgeon to the Adelaide Hospital, becoming Honorary Surgeon in 1925. The University of Adelaide appointed him Surgical Tutor and subsequently Lecturer in Clinical Surgery. His method of teaching was emphatic and clear. For several years he was Surgeon to the Repatriation Hospital. He was most zealous in his attendance and was always anxious to do all he could for the deserving soldiers. But woe betide the man who tried "to put it across him." An avalanche of wrath would descend on the culprit's head. Corbin was elected a member of the South Australian Branch of the British Medical Association in 1903 and took a prominent place in its activities for the rest of his life. He read several papers and showed many cases of interest at the scientific meetings. One of his first services was to visit Victoria and report on the working of the dispensary system at Geelong and Bendigo. His report was of the greatest value to the Branch Council. He was one of the first members of the Australasian Medical Publishing Company when that body absorbed the *Australasian Medical Gazette* and *Australian Medical Journal* in 1914. He was elected to the Council of the South Australian Branch of the British Medical Association in 1912 and became Vice-President in 1914. He was again elected to the Council in 1921. To his great pride he was elected President of the South Australian Branch in 1928, the jubilee year of its foundation, in which his father had played so notable a part. Those who were present at the Commemoration Conversazione will always remember the dignity and charm of the President and Mrs. Corbin. Other positions occupied by John Corbin were those of President of the Massage Association of South Australia and of the Adelaide Medical Students' Society. In the world of sport he had in his blood the Irishman's love of a horse and he was never loath to back his fancy. He was also very fond of golf and of fishing and shooting. John Corbin was a man of fine physique, handsome bearing and great personal charm. As a *raconteur* few could be so entertaining; his voice and gestures never made even a repetition boring. A man of impulsive explosive temper, it is not surprising that he had his differences, but he never bore a grude for long.

With his great capacity for friendship it is not surprising that his passing should leave many an aching heart. This was shown by the large attendance when his body was laid at rest in the beautiful little cemetery overlooking the scene of so much of his labours.

His wife was Miss Margaret Ogilvie, of Melbourne, who, with a son and two daughters, survives him. The sympathy of John Corbin's many friends will be with them in their sorrow.

H. S. Newland.

EDGAR HERBERT THANE.

WE regret to announce the death of Dr. Edgar Herbert Thane which occurred at Gordon, New South Wales, on March 12, 1930.

HENRY LYON JOHNSTON.

WE regret to announce the death of Dr. Henry Lyon Johnston which occurred at Perth, Western Australia, on March 5, 1930.

Books Received.

CLINICAL ATLAS OF BLOOD DISEASES, by A. Piney, M.D., M.R.C.P., and Stanley Wyard, M.D., M.R.C.P.; 1930. London: J. A. Churchill. Post 8vo., pp. 99, with illustrations. Price: 12s. 6d. net.

THE FEMALE SEX HORMONE, by Robert T. Frank, A.M., M.D., F.A.C.S.; 1930; London: Baillière, Tindall and Cox. Crown 4to., pp. 321, with illustrations. Price: 25s. net.

A REVIEW OF ARTIFICIAL LIGHT THERAPY SHOWING ITS PROVED VALUE IN MEDICAL AND SURGICAL PRACTICE, Compiled from authoritative and official sources and edited by R. King Brown, B.A., M.D.; D.P.H.; 1929. London: The Actinic Press, Limited. Demy 8vo., pp. 32. Price: 2s. net.

Diary for the Month.

MAR. 25.—New South Wales Branch, B.M.A.: Council.
MAR. 26.—Victorian Branch, B.M.A.: Council.
MAR. 27.—South Australian Branch, B.M.A.: Branch.
MAR. 28.—Federal Committee of B.M.A. in Australia.
MAR. 28.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. Henry Rogerson (B.M.A.) has been appointed Acting Medical Superintendent of the Hospital for the Insane, Kew, Victoria.

* * *

Dr. Henry Shannon (B.M.A.) has been appointed Certifying Medical Practitioner at Malvern, Victoria, pursuant to the provisions of the *Workers' Compensation Act*, 1928.

* * *

Dr. C. W. Anderson (B.M.A.) has been appointed Medical Officer of Health by the Dalwallinu Local Board of Health, Western Australia.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes*, sought, etc., see "Advertiser," page xviii.

GREEN PONDS MUNICIPALITY, KEMPTON, TASMANIA: Medical Officer of Health.

MARYBOROUGH HOSPITALS BOARD, QUEENSLAND: Junior Resident Medical Officer.

ROYAL NORTH SHORE HOSPITAL OF SYDNEY: Honorary Assistant Surgeon for Venereal Diseases.

THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Resident Medical Officer, Temporary Honorary Assistant Anæsthetist and Relieving Medical Officer.

TOOWOOMBA HOSPITALS BOARD, QUEENSLAND: Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 21, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting apopintments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Mount Isa Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia. Booleroo Centre Medical Club.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Medical practitioners are requested not to apply for appointments to positions at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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